TECHNICAL MEMORANDUM 3: NORTH SAN FRANCISCO BAY SELENIUM TOXICOLOGICAL ASSESSMENT

DRAFT

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ABBREVIATIONS

AET	apparent effects threshold
ANOVA	Analysis of variance
CDH	California Department of Health
DFG	Department of Fish and Game
dw	dry weight
EC01	concentration at which an effect is observed in 1% of the population (i.e., effect concentration 1%)
EC10	concentration at which an effect is observed in 10% of the population (i.e., effect concentration 10%)
LC50	concentration lethal to 50% of test subjects
LOAEL	lowest observed adverse effects level
MBTA	Migratory Bird Treaty Act
Na_2SeO_3	sodium selenite
NRC	National Research Council
NIWQP	National Irrigation Water Quality Control Program
NOAEL	no observed adverse effect level
Se	selenium
SeMet	selenomethionine
SFEI	San Francisco Estuary Institute
SMCV	Species mean chronic values are calculated by taking the geometric mean of the effect thresholds (e.g., NOAEL and LOAEL) for a particular species.
TMDL	total maximum daily load
UCL95	95% upper confidence limit on the mean
USDOI	U.S. Department of the Interior
USEPA	U.S. Environmental Protection Agency
USFWS	U.S. Fish and Wildlife Service

USGS	U.S. Geological Survey
ww	wet weight

1 INTRODUCTION

This technical memorandum provides a review of the available information on the toxicity of selenium to invertebrates, fish, birds, and marine mammals. This information has been developed to support the Regional Water Quality Control Board, San Francisco Bay Region (Water Board) in determining numeric targets for the selenium TMDL for the North San Francisco Bay (North Bay). The review of the selenium toxicity data was conducted in several steps. First, the available toxicity data from the published scientific literature and unpublished reports were compiled. These documents were identified from on-line searches of scientific references and the literature-cited sections of these reports were reviewed to identify unpublished reports. We estimate that greater than 95% of the relevant English-language literature has been reviewed. Next, the reported toxicity values were screened to identify those data that were most relevant to setting a numeric target for North Bay fish and wildlife. Screening criteria included:

- Chronic exposure to single chemical (selenium)
- Controlled experimental conditions
- Dietary exposure
- Tissue concentrations reported for exposed animals

The reported toxicity values (tissue concentrations) for individual species can exhibit a wide range due to differences in the corresponding toxicity endpoint of the test. Therefore, the test endpoint merits special attention in interpreting the reported toxicity values. The tables in this report, summarizing the selenium toxicity studies evaluated, report both the toxicity value and the test endpoint.

The focus of this report is on the compilation of relevant toxicity values. Some of the key issues to consider in interpreting the results of the laboratory tests and data analyses are also identified.

1.1 BACKGROUND INFORMATION

The current EPA and California state freshwater water chronic quality criterion for the protection of aquatic life (5 µg/L) has been criticized as inadequately representative of the toxic risks of selenium to aquatic life. Recent studies on selenium toxicity in aquatic food chains have generally reached the conclusion that a water-based criterion is not suitable due to "…temporal [and spatial] changes in concentrations, speciation, and rates of transfer between water, sediment and organisms…" (Hamilton 2004). Since the primary route of exposure to selenium is via the diet, and selenium is highly bioaccumulative, these differences can mean that a concentration of selenium in water that results in adverse effects in one location may not result in adverse effects to the same species in another location. Thus, the current recommendation (USEPA 2004, Chapman 2007, Hamilton 2002, 2004) for the appropriate media for regulation of selenium in the aquatic environment is not water, but rather tissue.

In 1987 in the North San Francisco Bay (hereinafter referred to as the "North Bay"), "…the California Department of Health (CDH) issued a health advisory for the consumption of three [sic] species of diving ducks in Suisun Bay, near San Francisco. These diving ducks (i.e., surf scoter, lesser and greater scaup) feed on bottom-dwelling animals (clams, mussels), which concentrate selenium from sediment and phytoplankton. In a study by the California Department of Fish and Game, the mean selenium levels in the muscle of duck were as follows: scoters, 3.6 ppm Se, wet weight; scaups, 2.2 ppm Se, wet weight. These levels were up to 5 times higher than mean Se levels in similar species in control areas (i.e., Humboldt Bay near the border of Oregon and California). The CDH recommended limiting the consumption of scoters to 4 oz/2 wk and of scaups to 4 oz/wk. Because of the known effects of selenium on the reproduction of birds, the CDH recommended that women of child-bearing age and children, 15 years old not consume these ducks" (Barceloux 1999).

In addition to the CDH health advisories for the consumption of diving ducks, the North Bay including Suisun Bay, Carquinez Strait, San Pablo Bay and Central Bay were listed in 1998, 2002, and 2006 under section 303(d) of the Clean Water Act due to impairments to both wildlife and human-health beneficial uses (Abu-Saba and Ogle 2002). It is on the basis of these listings that the Regional Water Quality Control Board, San Francisco Region (Water Board) is developing the Total Maximum Daily Load for the North Bay.

1.2 RELEVANT TOXICITY DATA

Several approaches have been used to develop screening values in the scientific and regulatory literature. These approaches include:

- LOAELs
- Effect thresholds
- Species mean chronic values
- EC01 or EC10
- Species sensitivity distributions

In the literature on selenium toxicity to fish, the approach that has generally been used is to set the screening value equal to the lowest LOAEL (i.e., the **lowest observed adverse effect level**) that has been reported (e.g., Hamilton 2003, 2004). When there is a large body of literature, with many reported LOAELs, this approach is likely to come close to the concentration at which effects first appear. However, when there are only a few studies, it is likely that effects begin at a level below the lowest LOAEL reported. For that reason, it may be advisable to use an SMCV, EC10, or effect threshold (see below) instead.

Effect thresholds are calculated as the geometric mean of the NOAEL (i.e., the highest **no observed adverse effect level**) and LOAEL reported for the same effect in an individual study. Since toxicity tests do not generally test many different concentrations, and effects may occur at concentrations below the LOAEL, calculating the geometric mean of the NOAEL and the LOAEL is one way to add a margin of safety to the LOAEL. That is, effects are likely to occur somewhere between the NOAEL and the LOAEL. The USEPA (1985, 2003) has made the decision that the geometric mean of the NOAEL and LOAEL is representative of where those effects are likely to occur.

Species mean chronic values (SMCVs) are used by USEPA in the calculation of water quality criteria (USEPA 1985). An SMCV is calculated by taking the geometric mean of the effect thresholds for a particular species. When there is only one study available, the results of that study are regarded as the SMCV.

Another approach that can be used to find the concentration at which effects are likely to occur between the NOAEL and LOAEL is to pool the data from similar studies and perform a regression (e.g., biphasic, hockey-stick, or logistic) on the response data. The data is generally only from a single species. This approach has been used in State of Utah (Ohlendorf 2007) by pooling the data from 6 studies on mallards and performing regressions on the concentration of selenium in feed and eggs vs. reproductive success. The State of Utah derived an EC10 (i.e., the **effect concentration 10%** or the concentration at which an effect is observed in 10% of the population) for selenium in mallard diets and eggs protective of reproductive success. This same approach can also be used to calculate an EC01 (i.e., **effect concentration 1%**), which will be lower, but protective of a greater proportion of individuals.

Species sensitivity distributions are another approach that have been used to derive screening values. In this approach, the probability distribution of some measure of toxicity (e.g., NOAEL, LOAEL, effect threshold, SMCV, LC_{50}, etc.) for multiple animal species is calculated. From the probability distribution, a screening value is then calculated that is protective of a certain proportion of the species (e.g., 95%). USEPA calculates percentiles of species sensitivity distributions for use in the derivation of water quality criteria (Posthuma et al. 2002). This approach is similar to the EC10 approach described above, except that it always uses data from multiple species, with each data point representative of a single species, and uses probability distributions instead of regressions to derive screening values. For the recent acute criterion for copper, USEPA compared their calculated value to a species sensitivity distribution (Figure 1-1) and found that it was protective of 95% of the genera making up the acute copper toxicity database (USEPA 2007a). The species sensitive distribution (Figure 1-1) contains the genus mean acute value (GMAV) for 27 genera, including 15 species of invertebrates, 22 species of fish, and 1 amphibian species. Each GMAV was calculated as the as the geometric mean of the species mean acute values (SMAV) for the species within that genera and the SMAV was calculated as the geometric mean of the individual value. The selected final acute value of 4.67 ug/L exceeds only one of 27 GMAVs (Figure 1-1); i.e., the GMAV for that cladoceran (an invertebrate) *Daphnia*.

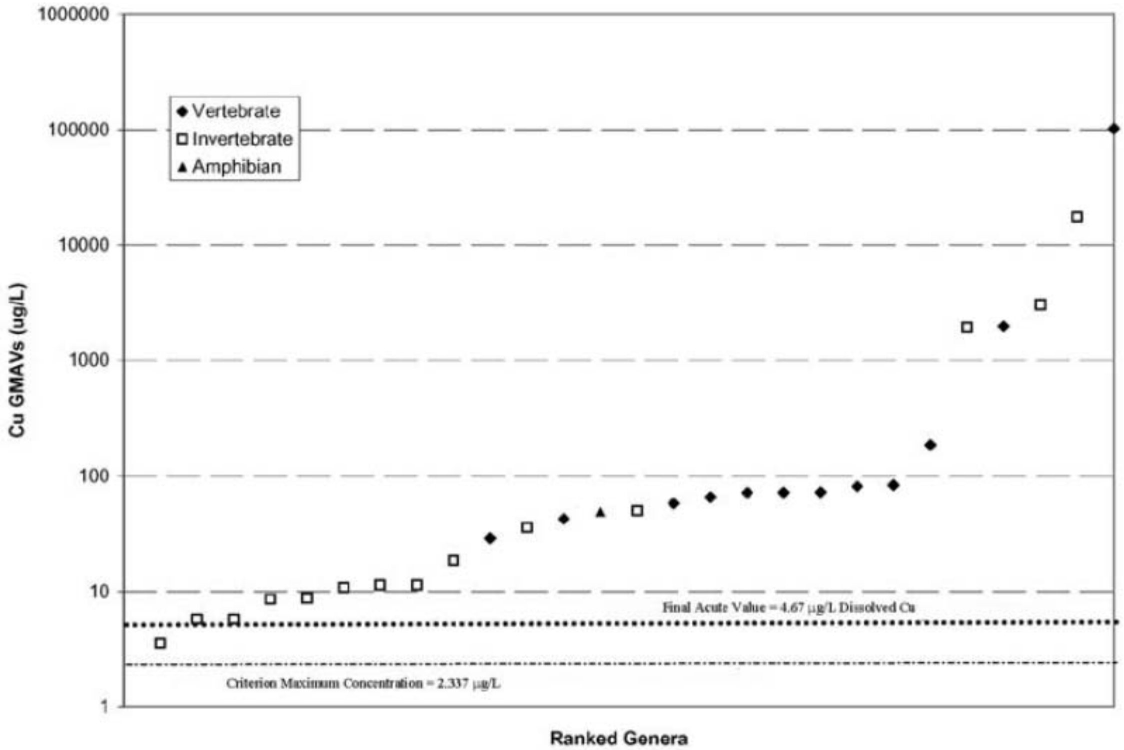

Figure 1-1 Ranked freshwater genus mean acute values (GMAVs) for copper (USEPA 2007a).

Effect thresholds and SMCVs will be calculated for the toxicity data presented in the following chapters. The lowest LOAEL will also be noted. Where an EC01, EC10, or species sensitivity distribution has been reported, that will also be noted. However, the calculation of these other measures of toxicity is not included in this review.

Selenium is an essential micro-nutrient. Adverse effects occur both when selenium concentrations are too low and too high in the diet (see Figure 1-2). At low concentrations, selenium is an essential micro-nutrient in animals. If the concentration of selenium in the diet is not adequate, selenium deficiency may result. The symptoms of selenium deficiency include edema, white muscle disease, reduced growth, reduced disease resistance, liver necrosis, morbidity, and increased mortality (National Research Council 1980, 2005, Ullrey 1992). At high concentrations, however, selenium can cause toxic effects, including emaciation, hair/feather loss, reduced reproductive success, reduced growth, developmental deformities, and mortality (National Research Council 1980, 2005, Ohlendorf 2003).

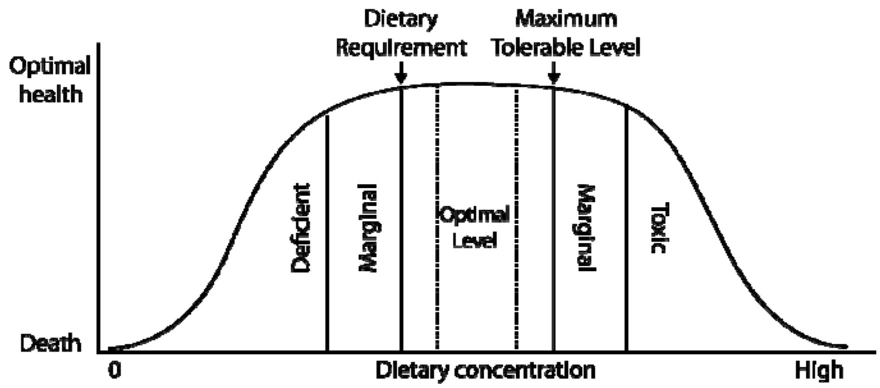

Figure 1-2 Health vs. dietary selenium concentration (adapted from NRC 2005).

The occurrence of effects in fish from low dietary selenium concentrations is seen in one of the toxicity studies examined in this report. In a study on fingerling channel catfish, Gatlin and Wilson (1984) were able to show that dietary selenium concentrations below approximately 0.3 mg/kg resulted in reduced weight gain. Above that, there was a plateau in the response (i.e., the optimal level) in which increased selenium concentrations did not have an adverse effect. However, the plateau ends at approximately 10 mg/kg, with concentrations above that also resulting in reduced weight gain (Figure 1-3). This has also been observed for calcium, magnesium, manganese, molybdenum, phosphorus, potassium, sodium, sulfur, and zinc (NRC 2005).

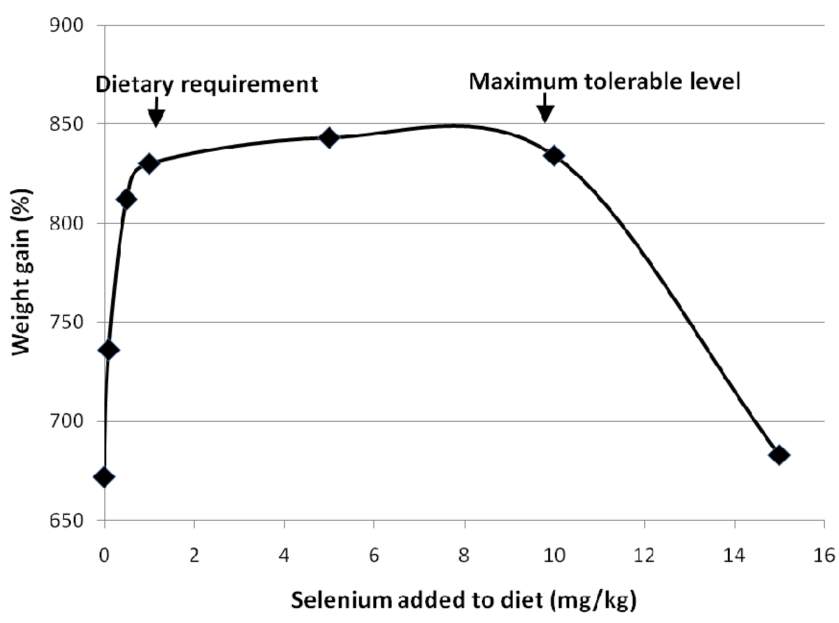

Figure 1-3 Weight gain in fingerling channel catfish as a function of dietary selenium (Gatlin and Wilson 1984).

Thus, screening values for dietary selenium concentrations should be set above the dietary requirement. When dietary concentrations are below the dietary requirements, adverse effects could theoretically occur from deficiency. Further, when dietary concentrations are near the requirements, a proportion of the population may also be adversely affected by selenium deficiency. In turn, these effects could lead to the very reduction in fish and bird populations that the selenium TMDL sought to avoid by reducing selenium concentrations. The dietary requirements of fish, birds, pigs, and other mammals have been determined by the NRC (1993, 1994, 1998) and will be presented in each of the sections evaluating the toxicity to the biota of the North Bay.

2 INVERTEBRATE TOXICITY REVIEW

Invertebrates are an integral part of many food chains in the North Bay, supporting many species of fish and birds, including species of recreational interest as well as threatened and endangered species. This suggests that if selenium negatively impacts the aquatic invertebrates in the North Bay, the result may be reduced food availability for the fishes and birds of the North Bay. Ultimately, this may lead to a reduction in the population sizes of those fishes and birds, even in the absence of direct impacts to the fishes and birds themselves from selenium. This suggests that the potential impacts of selenium on aquatic invertebrates in the North Bay should be part of the TMDL process.

While there is a large amount of literature on the toxicity of selenium to fishes, birds, and mammals, the chronic toxicity literature is very sparse for aquatic invertebrates. Thus, there is very little literature that may help the Water Board to determine whether the aquatic invertebrates in the North Bay are impacted by selenium. Similarly, the paucity of information on the toxicity of selenium to aquatic invertebrates means that are very few published sources of selenium screening values; e.g., no "Consensus-Based Sediment Quality Guidelines" (MacDonald et al. 2000), no California sediment quality objectives (SWRCB 2007), and only an apparent effect threshold in the NOAA SQuiRTs (Buchman 1999).

Despite the paucity of selenium toxicity data for aquatic invertebrates, there are a few chronic toxicity studies that may be informative to the Water Board. These studies are listed and briefly described below:

- Alaimo et al. (1994)
- Bielmeyer et al. (2005)
- Brasher and Ogle (1993)
- Debruyn and Chapman (2007)
- Hyne et al. (2002)
- Ingersoll et al. (1990)
- Jensen et al (2007)
- Malchow et al (1995)
- Naddy et al (1995)
- NOAA SQuiRTs (Buchman 1999)

Alaimo et al. (1994)

The authors performed chronic toxicity in a laboratory on the larvae of the midge *Chironomus decorus*. Chironomids are a major component of fish and waterfowl diet.

Midge eggs were placed in “moderately hard” water and, after hatching, were fed with powdered plants that were collected from evaporation ponds in California with varying concentrations of selenium. The authors found that increased selenium concentrations in the powdered food resulted in lower body weights of the growing midges with a NOAEL and LOAEL of approximately 0.5 and 1.6 mg/kg-dw, respectively, in the powdered food (effect threshold of 0.9 mg/kg-dw); the NOAEL and LOAEL for selenium in midge tissues were both below the detection limit of approximately 0.5 mg/kg-dw. In this experiment, selenium was only added via the food, but that also led to increases in selenium in the water column over time.

Bielmeyer et al. (2005)

The authors performed chronic toxicity tests in a laboratory on the larvae of sea urchins. Although the species tested, *Diadema antillarum*, does not occur in the North Bay, the authors state that “*sea urchin larvae have been shown to be highly sensitive to metals and therefore have been extensively used in marine pollution bioassays*” (Bielmeyer et al. 2005). Therefore, their results should be applicable to the North Bay in a general sense. The authors examined the development of larval sea urchins by exposing them to 0, 5, 10, 20, 36, 73 µg/L selenium as Na_2SeO_4 for 40 hours in sea water. The authors found that selenium cause abnormal development with an EC50 of 26 µg/L. However, there were no apparent adverse effects at up to 20 µg/l (i.e., the NOAEL was 20 µg/L). The experiments design that the authors used did not expose the parents to selenium, but only the larvae. In the North Bay, both the parents and the offspring will be exposed, which may potentially increase selenium sensitivity.

Brasher and Ogle (1993)

The authors performed chronic toxicity tests in a laboratory on the freshwater amphipod *Hyallela azteca*. Separate experiments were conducted using sodium selenite at 0, 50, 100, 200, 300, and 400 µg/L and sodium selenate at 0, 100, 250, 350, 500, and 700 µg/L for 24 days. Reproduction was reduced at 200 µg/L selenite but no effects were observed for selenate.

Debruyn and Chapman (2007)

The authors compiled existing toxicity data on major effects (i.e., toxic effects that are likely to affect a species at the population level, including reproduction and growth) that included water, dietary, or tissue selenium concentrations associated with toxic effects to 29 macroinvertebrate species. Studies reporting dietary and tissue concentrations were pooled across freshwater benthic, freshwater planktonic, and terrestrial invertebrate groups. For chronic waterborne exposures, the authors found that the amphipod *Hyallela* was the most sensitive with a median lethality reported at 100 µg/L selenite, 200 µg/L selenate, and a >50% reduction in reproduction at 100 µg Se/L selenate. Sublethal effects from dietary exposures were found to range from 1 to 80 mg/kg-dw. Sublethal effects for tissue concentrations of selenium were found to range from 1 to 30 mg/kg-dw. Although the authors used the data collected to construct a percentile based species sensitivity distribution (not a probability), they did not use the distributions to calculate values protective of a percentage of the species observed.

Hyne et al. (2002)

The authors performed chronic toxicity tests in a laboratory on juveniles and adults of the estuarine amphipod *Corophium* sp. Amphipods exposed to sediments spiked with seleno-L-methionine to give final concentrations of 0, 0.3, 1, 3, and 10 mg/kg-dw. Selenium was not added to either the water or the food given to the amphipods and all experiments were conducted using seawater. The only response measured in the amphipods was mortality. The results of these experiments are summarized in the Table 2-1 below (from Hyne et al. 2002).

Table 2-1
Summary of Results from Hyne et al. (2002)

Lifestage	Test conditions of overlying water	10-day LC_{50} sediment (dry wt) µg Se/g (95% CL)	10-day NOAEL sediment µg Se/g (dry wt)	10-day LOAEL sediment µg Se/g (dry wt)
Juvenile	No renewal	1.6 (1.3-1.9)	0.84	2.0
Juvenile	Daily renewal	6.3 (5.5-7.2)	1.7	5.0
Adult	No renewal	7.6 (6.8-8.4)	4.6	11.0

These results indicate that significant mortality was observed at concentrations as low as approximately 2 mg/kg-dw. An effect threshold was calculated for juvenile mortality in waters without renewal of 1.3 mg/kg-dw. However, it should be noted that since the authors only measured mortality, sublethal effects may be expected at lower sediment concentrations.

Ingersoll et al. (1990)

The authors performed chronic toxicity tests in a laboratory on the freshwater arthropods *Daphnia magna* and *Chironomus riparius*. Chironomids are a major component of fish and waterfowl diet. The experiments produced LOAELs for inorganic selenium in waterborne exposures of 85 µg/L for *Daphnia* and 837 µg/L for *Chironomus*.

Jensen et al. (2007)

The authors performed freshwater chronic toxicity tests in a laboratory on the larvae of the southern house mosquito *Culex quinquefasciatus. Culex* spp. are algal and bacterial feeders and are important as food for many organisms. The larvae were exposed to 2, 4, 8, 16, and 32 mg/L selenate. The experiments produced a LOAEL for reduced growth of 2 mg/L.

Malchow et al. (1995)

The authors performed freshwater acute toxicity tests in a laboratory on the larvae of the midge *Chironomus decorus.* Chironomids are a major component of fish and waterfowl diet. The experiments consisted of feeding the midge larvae with a diet of algae (*Selenastrum capricornutum*) cultured in three concentrations of selenite (0, 10, and 40 µg/L) or four concentrations of selenate (0, 4, 10, and 40 µg/L). The experiments produced a NOAEL and LOAEL for reduced body weight of 1.10 and 2.11 mg/kg-dw, respectively, in algae (effect threshold of 1.5 mg/kg-dw); or a NOAEL and LOAEL of <0.25 and 2.55 mg/kg-dw, respectively, in the midges (effect threshold of 0.8 mg/kg-dw, assuming the NOAEL was 0.25). Although this was an acute toxicity test, this is one of the few studies where invertebrates were exposed to selenium in the diet. Chronic tests may result in toxicity observed at lower concentrations.

Naddy et al (1995)

The authors performed freshwater chronic (9 day) toxicity tests in a laboratory using *Ceriodaphnia dubia*. The daphnia were exposed to <170, 510, 610, and 870 μg/L selenate. No effects on survival or reproduction were observed at any concentration.

NOAA SQuiRTs (Buchman 1999)

The National Oceanic and Atmospheric Administration has published a set of screening values called the Screening Quick Reference Tables (SQuiRTs). The SQuiRTs contain a marine sediment apparent effects threshold (AET) protective of amphipods of 1 mg/kg-dw. AETs are described as "…the concentration above which adverse biological impacts would always be expected… Conversely, adverse impacts are known to occur at levels below the AET… AET values were developed for use in Puget Sound (Washington)…"

2.1 DISCUSSION

"Selenium toxicity is greater in laboratory tests in which organisms are fed than in water only tests, because food accumulates Se from the aqueous phase, adding substantially to the test organism's total exposure" (Debruyn and Chapman 2007). Therefore, the results of water only tests can result in apparently high toxicity thresholds for invertebrates; e.g., 26 μg/L for sea urchins (Bielmeyer et al. 2005) and 2,000 μg/L for *Culex quinquefasciatus* (Jensen et al. 2007). Similarly, for sediment dwelling invertebrates, exposures to selenium in sediments appears to result in adverse effects at relatively low levels.

From the few studies presented above that have been performed using either dietary or sediment exposures, the following Table 2-2 was constructed:

Table 2-2
Effect thresholds for invertebrates exposed to dietary or sediment-bound selenium

	Exposure	Effect Threshold (mg/kg-dw)			
Organism	route	Diet	Sediment	Organism	Reference
midge	diet	0.9	-	<0.5	Alaimo et al. (1995)
amphipod	sediment	-	1.3	-	Hyne et al. (2002)
midge	diet	1.5	-	0.8	Malchow et al. (1995)

These data indicate that invertebrates may be affected by relatively low selenium concentrations, when exposed to selenium in dietary items or in sediments (for sediment dwelling biota).

Nutritional requirements

Selenium is a micro-nutrient (see Section 1). When animals do not have enough selenium in their diet, selenium deficiency results (NRC 1993, 1994). While the NRC (1993, 1994, 1998) has not established selenium nutritional requirements for invertebrates, it has established nutritional requirements for animals that potentially feed on invertebrates: 0.25-0.3 mg/kg for fish (NRC 1993), 0.17-0.33 for pigs (NRC 1998), and 0.16-0.22 mg/kg for birds (NRC 1994).

2.2 CONCLUSIONS

The effect thresholds shown in the table above are similar to the few published lower bound values available; i.e., the AET for selenium in marine sediments of 1 mg/kg is very close to the effect threshold of 1.3 mg/kg-dw observed by Hyne et al. (2002); and the lower bound dietary and organism selenium concentrations published by Debruyn and Chapman (2007) of 1 mg/kg-dw each are close to the effect thresholds reported for midges above (0.9-1.5 mg/kg-dw for diet and <0.5-0.8 mg/kg-dw for organism). All of these values are above the dietary requirements for fish, birds, and pigs (NRC 1993, 1994, 1998), although some are close.

Based upon the data presented here, there are several options for screening values protective of invertebrates. Those options are as follows (all in units of mg/kg-dw):

1. Dietary items
   a. Lowest effect threshold: 0.9
   b. Lowest LOAEL: 1.6
2. Sediment
   a. Lowest effect threshold: 1.3
   b. Lowest LOAEL: 2.0
   c. AET: 1.0
3. Organism (excluding non-detects)
   a. Lowest effect threshold: 0.8
   b. Lowest LOAEL: 2.55

3 FISH TOXICITY REVIEW

The North Bay supports a diverse fish biota. The fish supported include both sportfish and threatened and endangered fish species. The five most common sportfish in the North Bay are (SFEI 2000; listed in order catch frequency):

1. Striped bass (*Morone saxatilis*)
2. Halibut (*Paralichthys californicus*)
3. Jacksmelt (*Atherinopsis californiensis*)
4. White sturgeon (*Acipenser transmontanus*)
5. White croaker (*Genyonemus lineatus*)

In addition to the sportfish listed above, the North Bay supports the following threatened and endangered fishes (USFWS 2007):

1. Chinook salmon (*Oncorhynchus tshawytscha*)
2. Delta smelt (*Hypomesus transpacificus*)
3. Green sturgeon (*Acipenser medirostris*)
4. Longfin smelt (*Spirinchus thaleichthys*)
5. Sacramento perch (*Archoplites interruptus*)
6. Sacramento splittail (*Pogonichthys macrolepidotus*)
7. Steelhead trout (*Oncorhynchus mykiss*)
8. Tidewater goby (*Eucyclogobius newberryi*)

Selenium in the North Bay may adversely impact these fish by causing reduced production of viable eggs, post-hatch mortality, deformities in growing larvae, and various pathological effects in the kidneys, liver, heart, and ovaries (Hamilton 2003, 2004; Lemly 2002a). These identified effects may lead to population declines of both the sportfish and the threatened and endangered fishes in the North Bay. Therefore, as part of the selenium TMDL for the North Bay, selenium screening values will be developed that are protective of the species listed above.

As stated in the Introduction (see Section 1), the potential screening values that are proposed here are tissue concentrations. For fish, measuring the concentration of selenium in dietary items poses a logistical challenge. Mainly, the challenge lies in that many fish feed on a wide array of small prey items. Collecting the right kind of prey items, and a sufficient number for analysis, can be very time consuming. Instead, it is generally recommended that selenium be measured in the fish of concern (Hamilton 2002, 2003, 2004, USEPA 2004). Additionally, it is recommended that these measurements be made in whole fish due to "*…practical reasons of sampling and because a sufficient data base containing chronic effects based on whole-body tissue is present in the literature*" (USEPA 2004). The amount of data for chronic effects based on other tissue types (e.g., ovary, liver, kidney , and muscle) is not as extensive and, therefore, not amenable to deriving screening values. Further, although "*ovaries may be the best tissue to link selenium to reproductive effects…*

ovarian tissue is also only available seasonally and sometimes difficult to extract in quantities sufficient for analysis…" (USEPA 2004). Therefore, whole body concentrations in fish are recommended for use as screening values.

3.1 METHODS

Selenium toxicity data for the fishes that occur in the North Bay would be the most relevant for developing selenium fish tissue screening values for the North Bay. However, for the fishes in the North Bay, there is very limited toxicity data and almost no toxicity data for fishes at all in the salinity range that occurs in the North Bay; i.e., 1 to 33.5 ppt. Therefore, this review is largely limited to the evaluation of the freshwater toxicity literature.

Selenium toxicity studies on fishes were gathered from the scientific literature, as well as reports and other "grey literature." The studies collected were evaluated to select those that provided fish tissue concentrations at which toxic effects from chronic selenium exposures were observed. To ensure that screening values were not influenced by other variables (e.g., other contaminants), only studies with controlled experimental designs were selected.

The criteria used to identify studies with usable toxicity information are as follows:

A. provide a NOAEL and/or a LOAEL

B. chronic

C. exposed to selenium only

D. only treatment was selenium exposure level

E. contained tissue data

F. fish (and their food) were raised in the laboratory under controlled conditions

G. used a dietary exposure scenario; mesocosm experiments in which selenium was added to the water column meet this requirement

All studies that were reviewed are listed in Table 3-1. Studies that were determined to be unsuitable are noted in Table 3-1 using the designations from the list above.

Table 3-1
Selenium toxicity studies evaluated in this review. Studies that were excluded from review are indicated (see text above for an explanation of the criteria).

Study	Reference	Evaluation criteria failed		Study	Reference	Evaluation criteria failed
1	Bennett et al. (1986)	A		41	Hilton and Hodson (1983)	
2	Bertram and Brooks (1986)	A		42	Hodson et al. (1980)	E
3	Brandão et al. (1992)	A		43	Hodson et al. (1986)	A
4	Chapman (1992)	B		44	Holm et al. (2005)	F
5	Cleveland et al. (1993)			45	Hunn et al (1987)	G
6	Coughlan and Velte (1989)	F		46	Kennedy et al. (2000)	F
7	Coyle et al. (1993)			47	Kimball (1978)	E
8	de Rosemond et al. (2005)	A		48	Klaverkamp et al. (1983)	E
9	Dobbs et al. (1996)			49	Kleinow (1984)	A
10	Doroshov et al. (1992)			50	Kleinow and Brooks (1986a)	A
11	Finger and Bulak (1988)	C,D		51	Kleinow and Brooks (1986b)	A
12	Finley (1985)	F		52	Lawler et al. (1981)	E
13	Gatlin and Wilson (1984)			53	Lemly (1993a)	
14	Gillespie and Baumann (1986)	F		54	Lemly (1993b)	F
15	Gissel-Nielsen and Gissel-Nielsen (1978)	E		55	Linville (2006)	
16	Goettl and Davies (1977)	E		56	Mehrle et al. (1982)	C,D
17	Goettl and Davies (1978)	E		57	Muscatello et al. (2006)	F
18	Hall et al. (1984)	C		58	Ogle and Knight (1989)	
19	Hall et al. (1985)	B,C,D		59	Pyron and Beitinger (1989)	A
20	Hall et al. (1987)	B,C,D		60	Saiki et al. (1992)	E
21	Halter et al. (1980)	B		61	Saiki et al. (2004)	C
22	Hamilton et al. (1986)	C		62	Schultz and Hermanutz (1990)	
23	Hamilton et al. (1990)			63	Schlenk et al. (2003)	B
24	Hamilton and Wiedmeyer (1990)	C		64	Sorensen and Bauer (1983)	E
25	Hamilton et al. (2000)	C		65	Sorensen and Bauer (1984)	A
26	Hamilton et al. (2002a)	C		66	Sorensen et al. (1982a)	A,E
27	Hamilton et al. (2002b)	C,D		67	Sorensen et al. (1982b)	A
28	Hamilton et al. (2005a)	C,D		68	Sorensen et al. (1983)	A
29	Hamilton et al. (2005b)	C		69	Sorensen et al. (1984)	F
30	Hamilton et al. (2005c)	C		70	Sorensen (1988)	F
31	Hamilton et al. (2005d)	C		71	Tashjian et al. (2006)	
32	Harrison et al. (1990)	A		72	Tashjian et al. (2007)	E
33	Hardy (2003)	F		73	Teh et al. (2002)	B,E
34	Hartwell et al. (1987a)	A		74	Teh et al. (2004)	
35	Hartwell et al. (1987b)	A		75	Vidal et al (2005)	
36	Hermanutz (1992)	E		76	Watenpaugh and Beitinger (1985a)	A
37	Hermanutz et al. (1992)			77	Watenpaugh and Beitinger (1985b)	B
38	Hicks et al (1984)			78	Watenpaugh and Beitinger (1985c)	A
39	Hilton et al. (1980)			79	Woock et al. (1987)	E
40	Hilton et al. (1982)	A		80	Wise et al. (1993)	E

The reported effects from each study were grouped into one of two categories: major and minor effects. Major effects are those that have the potential to impact fish at the organism and/or population level (e.g., increased mortality, reduced fecundity, reduced growth, etc.). Minor effects are those that are measurable, but are unlikely to result in population level effects and/or the long-term impacts on individual fish performance was unclear (e.g., a change in an enzyme concentration). Where both major and minor effects were observed at the same concentration, the effects was assumed to be major. Only major effects were considered in development of screening values.

Effect thresholds were calculated as the geometric mean of the NOAEL and LOAEL reported for the same effect in an individual study, as is recommended in USEPA water quality criteria development guidance (USEPA 1985). Additionally, species mean chronic values (SMCVs) were calculated as the geometric mean of the effect thresholds (USEPA 1985). Studies that did not report a NOAEL were excluded from the calculation of an effect threshold and the SMCV. Note that excluding studies in which a NOAEL was not reported from the calculation of the SMCV results in lower SMCVs. USEPA (1985) provides the following rationale for the use of geometric means to calculate SMCVs:

"Geometric means rather than arithmetic means are used here because the distributions of individual organisms' sensitivities in toxicity tests on most materials, … are more likely to be lognormal than normal."

The calculated SMCVs can then be used to evaluate the relative sensitivity of each species of fish to selenium and help determine the appropriate threshold for use.

In studies where selenium was not measured in whole fish (e.g., selenium was measured in liver or muscle), measured concentrations were converted to whole body concentrations using the equations presented by USEPA (2004). Additionally, tissue values based upon wet-weight values were converted to dry-weight values using either the %moisture values presented in the same study or default values from USEPA (2004). All conversion methods are described in detail in Appendix A.

3.2 Results

The toxicity studies from Table 3-1 that were included in the evaluations below included 18 studies on the following fish species:

- Bluegill
- Channel catfish
- Chinook salmon
- Fathead minnows
- Rainbow trout
- Sacramento splittail
- White sturgeon

Bluegill

Five suitable studies were found on bluegills. Whole body effect thresholds ranged from 3.0 to 13.8 mg/kg-dw (Table 3-3).

Channel catfish

Two suitable studies were identified; i.e., Doroshov et al. (1992) and Gatlin and Wilson (1984). In the study by Doroshov et al. (1992), the exposure route was intramuscular injection of selenomethionine. The degree of correlation between intramuscular injection and dietary exposures for selenium is unknown. In the study by Gatlin and Wilson (1984), selenium exposure was dietary. From these studies, we calculated whole body effect thresholds of 2.7 and 3.3 mg/kg-dw, respectively (Table 3-3).

Chinook salmon

A single suitable study was identified (Hamilton et al. 1990). That study evaluated selenium toxicity in both brackish and freshwaters, the whole body effect thresholds calculated from this study were 7.6 mg/kg-dw for freshwater and 17.1 mg/kg-dw for brackish water (Table 3-3) for fishes fed SeMet. These results indicate that selenium may be less toxic to salmon in brackish than in fresh waters.

Beckon (2007) also analyzed the data from Hamilton et al. (1990) but included the data from salmon that were fed field-collected mosquitofish. In our analyses, we excluded the data from salmon fed mosquitofish caught in the field as this exposed the salmon to multiple contaminants simultaneously which might have negatively influenced survival. Instead of calculating an effect threshold from the LOAEL and NOAEL from Hamilton et al. (1990), Beckon (2007) performed a regression of selenium concentration vs. survival and estimated an EC10 and EC20 of 1.84 and 2.5 mg/kg-dw, respectively. The analysis in shown in the figure below.

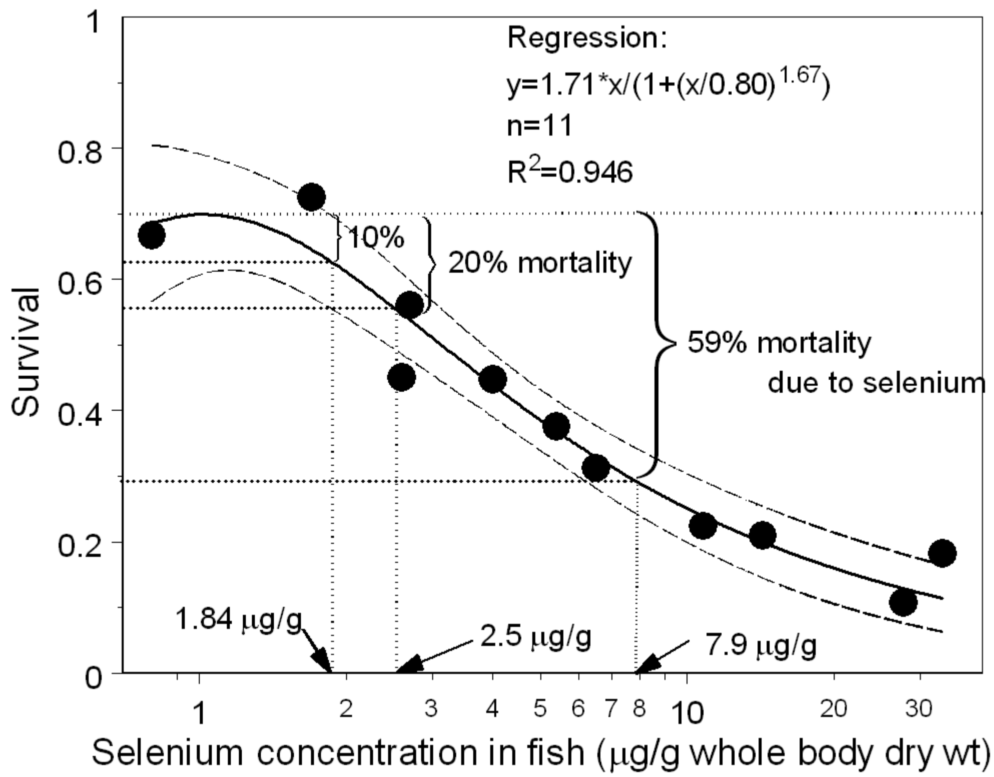

Figure 3-1 Biphasic regression analysis of the data from Hamilton et al. (1990)

It should be noted that performing a regression on the treatment means, instead of the raw data from individual test organisms, masks the true variation associated with the experimental data; i.e., the confidence limits associated with the regression line in Figure 3-

1 could be much larger. Further, the nature of the relationship between fish survival and whole-body selenium concentrations (i.e., the EC10) may change when using the raw data. To examine the effects of using the data for the individual test organisms in the analysis, we used the treatment means and standard errors for the same 11 treatments that Beckon (2007) used to create the regression analysis in Figure 3-1 and repeated the experiment 10 times in a Monte Carlo simulation analysis. Assuming a normal distribution, we generated 2 data points per treatment (i.e., the number of data points collected by Hamilton et al. (1990)) and performed the same regression analysis. An example graph from one of the simulation experiments is provided in Figure 3-2.

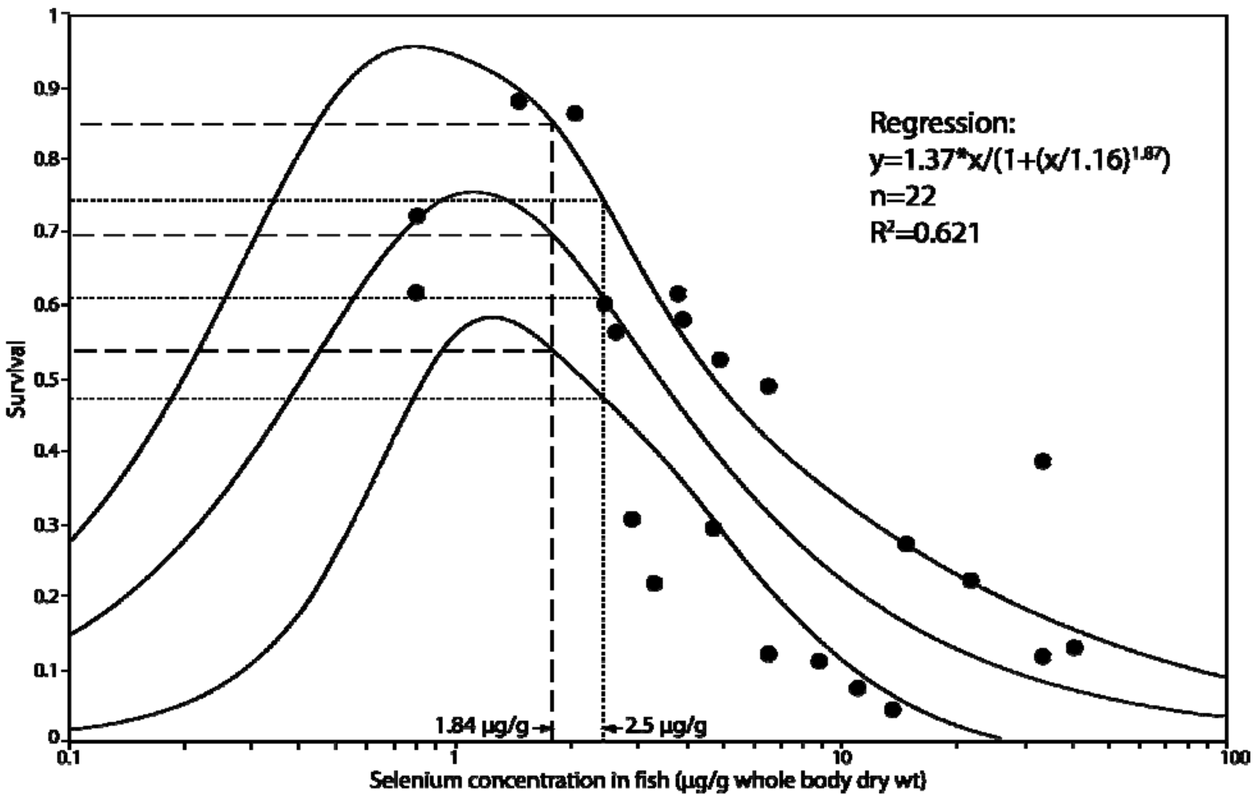

Figure 3-2 Biphasic regression based on simulated data from Hamilton et al. (1990)

From this simulated data set (Figure 3-2), it can be seen that performing a regression analysis on the (simulated) raw data (vs. the treatment means) leads to wider confidence intervals on the regression line. For example, the confidence limits on the regression line at the lowest treatment level are approximately 48-97% survival, as opposed to the approximately 57-80% survival that was calculated using only the treatment means.

The EC10 and EC20 derived from all 10 simulated experiments are presented in Table 3-2. The graphically derived confidence limits are also presented.

Table 3-2
The EC10 and EC20 from 10 simulated experiments and 95% confidence limits (CLs) on salmon survival at the EC10 and EC20

EC10				EC20		
(mg/kg-dw)	LCL for salmon survival at EC10[a]	UCL for salmon survival at EC10[a]		(mg/kg-dw)	LCL for salmon survival at EC20[a]	UCL for salmon survival at EC20[a]
1.91	52	83		2.47	47	73
2.03	55	88		2.61	49	79
1.59	57	76		2.38	50	68
1.82	51	74		2.50	46	70
1.60	47	72		2.26	41	64
1.88	46	71		2.52	41	64
1.82	50	77		2.46	45	68
1.94	48	75		2.74	43	67
2.42	55	72		3.33	49	64
1.85	49	74		2.65	44	66

Notes:
a - Confidence limits are graphically derived
UCL – upper confidence limit
LCL – lower confidence limit

Further, from Figure 3-2 and Table 3-2, it appears that the EC10 and the EC20 derived by Beckon (2007) may not significantly different from each other and that both may not significantly different from the no effect level in the regression; i.e., 70% survival. Testing for significant differences among the no effect level, the EC10, and the EC20 requires the ability to set 95% confidence limits. For non-linear regression analyses, the likelihood-ratio method is recommended for calculating confidence limits (see Moerbeek et al. 2004, Sand et al. 2006). However, we have not yet taken this next step to calculate confidence limits on the EC10 and EC20.

Fathead minnows
Three suitable studies were identified for fathead minnows. Whole body effect thresholds ranged from 6.0 to 73 mg/kg-dw (Table 3-3).

Rainbow trout
Four suitable studies were identified for rainbow trout (Table 3-3). Whole body effect thresholds for selenium ranged from 1.7 to 53.1 mg/kg-dw (Table 3-3). Using the treatment means from one of the suitable studies (i.e., Hilton et al. 1980; see study 38 in Table 3-3) Beckon (2007) derived an EC20 for juvenile rainbow trout of 2.15 mg/kg-dw based on a 20% reduction in body weight. However, this approach is subject to the same limitations discussed above for chinook salmon.

It should also be noted that Beckon (2007) used the raw data from a study that did not meet our selection criteria (i.e., Holm et al. 2005) to derive an EC20 of 2.93 mg/kg-dw for adult rainbow trout, based on a 20% reduction in the survival of fry from tout exposed to selenium.

Sacramento splittail
A single suitable study was identified (Teh et al. 2004). In that study, an effect threshold of 12.3 mg/kg-dw (muscle) was determined. Using the equations in USEPA (2004) to convert

muscle concentrations to whole body concentrations, an effect threshold of 10.8 mg/kg-dw (whole body) was calculated (Table 3-3).

White sturgeon

Two suitable studies were identified which evaluated the toxicity of selenium to white sturgeon (i.e., Linville 2006, Tashjian et al. 2006). Linville (2006) performed two sets of experiments: 1) injecting selenium into sturgeon eggs and 2) exposing adult sturgeon to dietary selenium. Only the results from the dietary exposure experiment are used in the evaluations presented here. We calculated whole body effect thresholds of 6.2 mg/kg-dw from Linville (2006) and 18.2 mg/kg-dw from Tashjian et al. (2006) (Table 3-3). William Beckon (2008) of the USFWS analyzed the data in Linville (2006) and derived an EC10 for selenium in muscle tissue which he then converted to a whole body concentration of 7.50 mg/kg-dw using a linear regression relating the two tissue types. The regression that Beckon (2008) used to convert selenium muscle concentrations to whole body concentrations was derived from the treatment means in Tashjian et al. (2006). Tetra Tech obtained the raw data from Tashjian et al. (2006) and used that to derive a linear regression relating muscle to whole body concentrations (see Appendix A). Using that regression, Tetra Tech converted the muscle tissue EC10 derived by Beckon (2008) into a whole body concentration of 11.9 mg/kg-dw. However, it should be noted that there were few data points available for the calculation of the EC10, making this value somewhat uncertain. The regression derived from the raw data from Tashjian et al. (2006) was also used to convert Linville’s (2006) muscle NOAEL and LOAEL into whole body concentrations (Table 3-3).

Table 3-3
Summary of selenium toxicity studies evaluated in this review.

Fish	Study	Water Type	Life stage	Liver[1] NOAEL (mg/kg-dw)	Liver[1] LOAEL (mg/kg-dw)	Muscle[1] NOAEL (mg/kg-dw)	Muscle[1] LOAEL (mg/kg-dw)	Whole Body NOAEL (mg/kg-dw)	Whole Body LOAEL (mg/kg-dw)	Effect Threshold (mg/kg-dw) Liver	Effect Threshold (mg/kg-dw) Muscle	Effect Threshold (mg/kg-dw) Whole Body	Endpoint
bluegill	5	fresh	Juvenile	16.8	24.5	5.1	8.4	4.7	7.6	20.3	6.5	6.0	BW, L
bluegill	7	fresh	Adult	30.1	49.1	11.1	21.5	10.0	19.0	38.4	15.4	13.8	R
bluegill	10	fresh	Adult	12.3	26.0	5.8	10.4	5.5	9.3	17.9	7.8	7.1	D
bluegill	37	fresh	Adult	6.0	37.2	1.2	7.2	1.3	6.7	14.9	2.9	3.0	BW, R, S
bluegill	53	fresh	Juvenile	6.3	19.9	1.4	6.3	1.3	7.8	11.2	2.9	3.2	S, BW, L
channel catfish	10	fresh	Adult	7.3	12.5	2.1	3.8	2.0	3.6	9.6	2.8	2.7	S
channel catfish	13	fresh	Fingerling	-	-	3.0	3.5	3.1	3.5	-	3.3	3.3	BW
chinook salmon	23	fresh	Larvae[2]	18.8	31.9	5.9	12.0	5.4	10.8	24.5	8.4	7.6	BW, L
chinook salmon	23	brackish	Juvenile[2]	35.9	57.2	14.1	26.4	12.6	23.2	45.3	19.3	17.1	BW, L, S
fathead minnow	9	fresh	Larvae	-	121.0	-	72.8	-	62.0	-	-	-	BW
fathead minnow	58	fresh	Juvenile	18.8	21.9	5.7	6.6	5.4	6.6	20.3	6.1	6.0	BW
fathead minnow	62	fresh	Egg	-	-	-	-	1.6	19.6	-	-	5.5	D
rainbow trout	75	fresh	Larvae	6.1	9.9	1.3	2.5	1.2	2.3	7.8	1.8	1.7	BW, L
rainbow trout	38	fresh	Juvenile	153.2	197.2	-	-	46.8	60.3	173.8	-	53.1	BW, FG, S
rainbow trout	39	fresh	Juvenile	42.0	95.0	-	-	12.6	28.9	63.2	-	19.1	BW, S, FG
rainbow trout	41	fresh	Juvenile[3]	21.0	71.7	1.0	4.3	1.0	4.0	38.8	2.1	2.0	BW, FG
Sacramento splittail	74	fresh	Juvenile	23.0	26.8	10.1	15.1	9.0	12.9	24.8	12.3	10.8	D
white sturgeon	55	fresh	Adult	1.4	10.4	1.3	12.1	3.1	12.3	3.9	4.0	6.2	D
white sturgeon	71	fresh	Juvenile	22.0	37.4	22.9	36.8	14.7	22.5	28.7	29.0	18.2	BW, SA

Notes:
1 - shaded cells represent concentrations predicted from measurements in other tissues (see Appendix A)
2 - only results from the SeMet dietary exposure part of the study are used here
3 - data from the low carbohydrate diet were not used as the authors reported that reduced weight of fish on this diet was likely due to food avoidance, not toxicity
BW = Body Weight
D = Deformities
FG = Feed:Gain ratio
L = Length
R= Reproduction
S = Survival
SA = Swimming Activity

Using the data presented in Table 3-3, SMCVs were calculated for each fish species. For species where only one toxicity study was reported, the SMCV is equal to the effect threshold from that study. The SMCVs are shown in the Table 3-4.

Table 3-4
Species mean chronic values calculated from the toxicity studies evaluated above.

Fish	Species	Water Type	SMCV (mg/kg-dw)		
			Liver	Muscle	Whole body
Bluegill	*Lepomis macrochirus*	fresh	18.8	5.8	5.6
Channel catfish	*Ictalurus punctatus*	fresh	9.6	3.0	3.0
Chinook salmon	*Oncorhynchus tshawytscha*	fresh	24.5	8.4	7.6
Chinook salmon	*Oncorhynchus tshawytscha*	brackish	45.3	19.3	17.1
Fathead minnow	*Pimephales promelas*	fresh	20.3	6.1	6.0
Rainbow trout	*Oncorhynchus mykiss*	fresh	42.7	1.9	7.6
Sacramento splittail	*Pogonichthys macrolepidotus*	fresh	24.8	12.3	10.8
White sturgeon	*Acipenser transmontanus*	fresh	10.5	10.8	10.6

Note: shaded cells are based (at least partly) on regressions, not measurements

Life History differences

To determine whether there is a trend in the sensitivity to selenium over the lifespan of a fish, the data gathered were segregated by life-history stage (e.g., adult, juvenile/fingerling, fry/larvae, egg). There were little data for fish larvae/eggs, and the majority of the available data are for effects on juveniles and adults. From the available data (Figures 3-3, 3-4) there does not appear to be a relationship between age and selenium sensitivity. A relationship may be present, but there is insufficient data available to adequately evaluate the existence of a pattern.

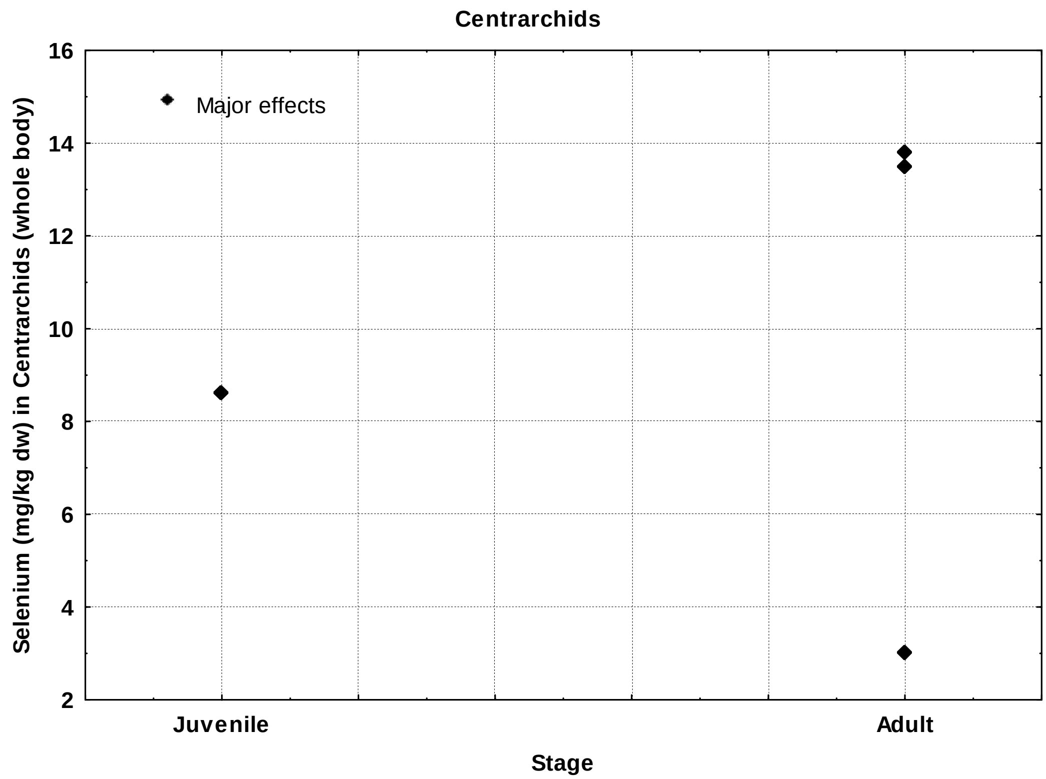

Figure 3-3 **Effect thresholds for centrarchids (i.e., bluegill, green sunfish, and redear sunfish) from the selected studies grouped by life history stage.**

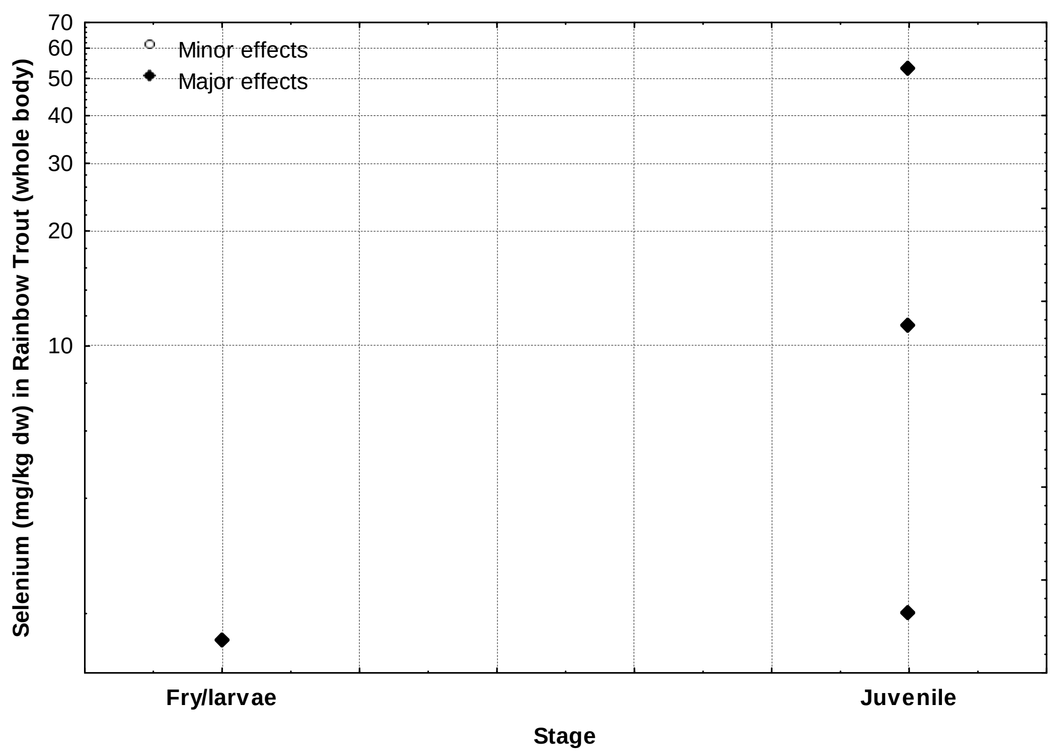

Figure 3-4 **Effect thresholds for rainbow trout from the selected studies grouped by life history stage.**

Since there were too few suitable studies to evaluate the potential effect of life history stage on selenium toxicity to fish, some of the criteria that had been used to exclude studies were relaxed. Specifically, criteria F and G of Table 3-1 were relaxed to increase our ability to evaluate life history stage. However, this means that studies that were included where fishes were either fed food that was not reared under controlled conditions or where fishes were exposed to selenium in water only. The results are shown in Figures 3-5 and 3-6. Note that the studies that were added by relaxing the selection criteria were not included in the calculation of SMCVs or screening values.

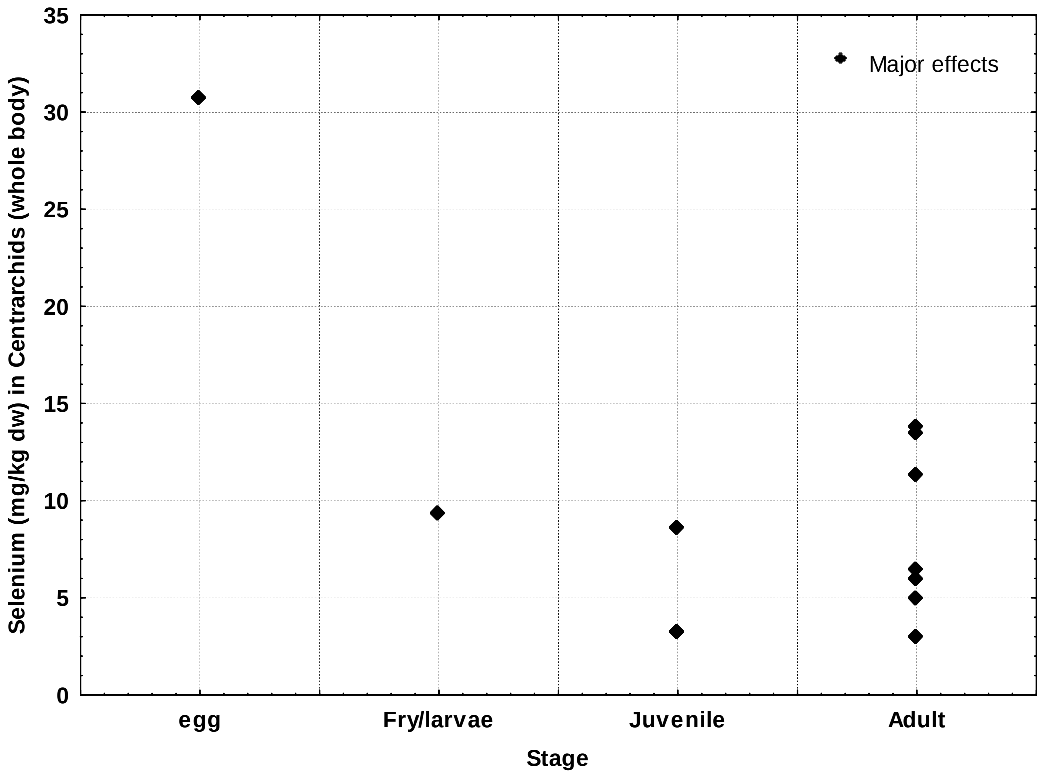

Figure 3-5 Effect thresholds for centrarchids after relaxing the selection criteria.

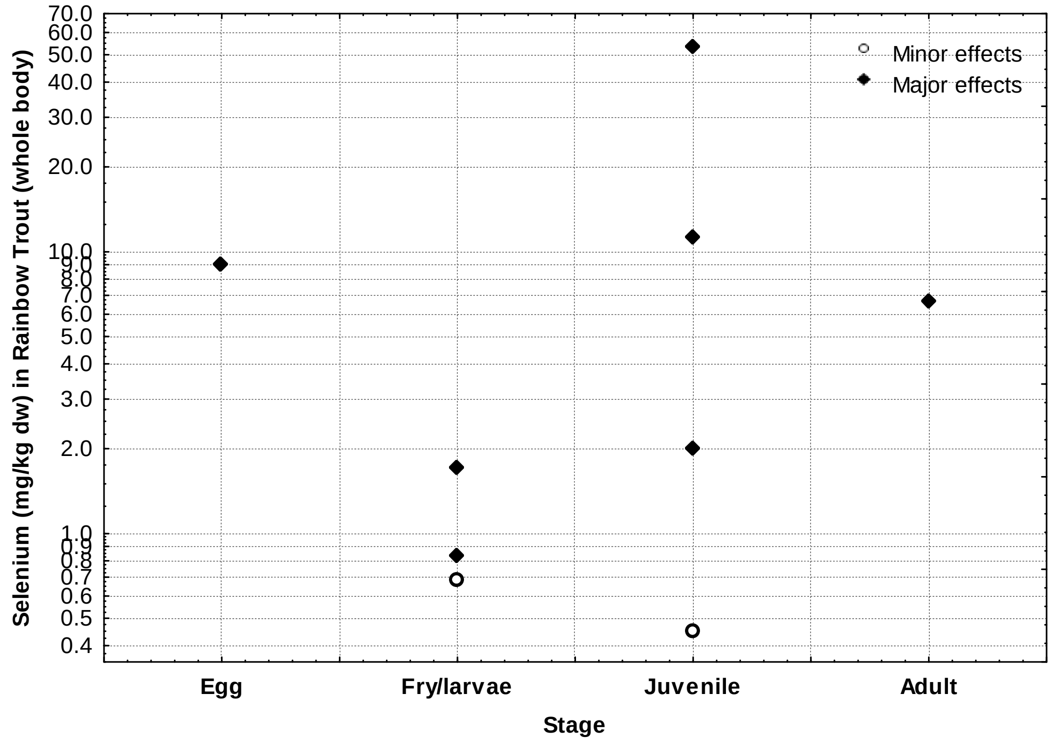

Figure 3-6 Effect thresholds for rainbow trout after relaxing the selection criteria.

Although the range of effect thresholds in Figure 3-5 overlap, the mean effect threshold for juvenile centrarchids is 1.7 times lower than for adults. No trend was apparent in the data available for rainbow trout. Overall, it does not appear that eggs are more sensitive than juvenile or larval fish. There are several potential reasons that the available data do not show that eggs are more sensitive than juvenile/larval fish: 1) the endpoints measured were not sensitive enough, 2) the type of selenium exposure was not conducive to observing effects on eggs (e.g., waterborne exposures), or 3) fish eggs may indeed be less sensitive to toxic chemicals than other life history stages (Finn 2007).

3.3 Discussion

80 selenium toxicity studies were reviewed in an effort to derive selenium screening values for species representative of fishes in the North Bay (Table 3-1). Based on this review, species which are appropriate for use in developing a fish tissue screening values for the North Bay, and for which usable selenium toxicity data are available, include: bluegill (*Lepomis macrochirus*), fathead minnow (*Pimephales promelas*), rainbow trout (*Oncorhynchus mykiss*), Chinook salmon (*Oncorhynchus tshawytscha*), Sacramento splittail (*Pogonichthys macrolepidotus*), and white sturgeon (*Acipenser transmontanus*).

With the exception of the study on the impacts of selenium on Chinook salmon in fresh and brackish water (Hamilton et al. 1990), all usable studies performed to date on the effects of selenium on fish have been performed in freshwater. In addition to these species, selenium toxicity data was sought for striped bass, flounder, and delta smelt. However, no usable selenium toxicity data were available for these fish, nor were data available for any other closely related species (e.g., pond smelt, *Hypomesus olidus*).

Based on the available data, it appears that the species of concern in the North Bay for which toxicity data are available (i.e., Sacramento splittail and white sturgeon) are less sensitive to selenium than bluegill (Table 3-4), a species that has been observed to be particularly sensitive to selenium in a number of studies (e.g., Lemly 1993a) and was selected by USEPA as the basis of the current draft water quality criterion for selenium (USEPA 2004). Further, channel catfish appear to be even more sensitive to selenium than bluegill. However, with only one dietary and one injection toxicity study available on catfish, confidence in the sensitivity of channel catfish to selenium is limited.

Other selenium screening values

The selection of an appropriate selenium fish tissue screening value, protective of toxic effects in both individual fish and populations, is an area of active debate (Chapman 2007; Hamilton 2003, 2004; Lemly and Skorupa 2007; McDonald and Chapman 2007; Skorupa et al. 2004; USEPA 2004). Numerous screening values for selenium in fish tissues have been proposed in the past. These values are summarized in Table 3-5.

Table 3-5
Whole body fish tissue screening levels (mg/kg-dw) published in the scientific literature.

Reference	No effect[a]	Level of concern[b]	Toxicity Threshold[c]
Lemly and Smith (1987)			12
Henderson et al (1995)	4	4-12	12
Stephens et al. (1997)	2-3	4	4
USDOI (1998)	2-3	2-4	4
DeForest et al (1999)			6 (cold water)
DeForest et al (1999)			9 (warm water)
Lemly (1993c, 1996, 2002b)			4
Hamilton (2003)			4
Ohlendorf (2003)			4
USEPA (2004)			7.9

Notes:
a - Concentrations less than this value produce no discernible adverse effects on fish or wildlife and are typical of background concentrations in uncontaminated environments (USDOI 1998).
b - Concentrations in this range rarely result in discernible adverse effects on some fish or wildlife species (USDOI 1998).
c - Concentrations greater than this value may result in adverse effects on some fish or wildlife species (USDOI 1998).

The earliest screening values were 12 mg/kg-dw (see Lemly and Smith (1987), Henderson et al. (1995)) (Table 3-5). However, as more data became available, most authors generally recommended a screening value of 4 mg/kg-dw (Table 3-5). In part, those screening values were derived to be protective of the lowest concentration at which selenium exposures resulted in toxicity in a single study. Thus, the approach used to derive the screening values in the scientific literature has generally been to use the lowest whole body values from Table 3-3. Further, the approach in the scientific literature has been to derive a screening value protective of all fish species across the entire United States and not just the North Bay.

There have also been several critiques of the USEPA (2004) draft that have noted statistical and interpretational errors in the draft that potentially affect the draft screening value (Lemly and Skorupa 2007, Skorupa et al. 2004).

Presser and Luoma (2006) also reviewed the toxicity literature and concluded that whole-body fish tissue concern levels range from 1.5 to 6 mg/kg-dw.

Warm water vs. cold water screening values

One of the major topics of discussion in the recent past has been whether cold water fish are more sensitive to selenium that warm water fish. Recent reviews have not found support for the cold vs. warm water distinction (e.g., Hamilton 2003, Chapman 2007).

Effect of sulfate on selenium toxicity

Acute toxicity tests have shown that sulfate reduces the potential for bioaccumulation and toxicity of selenium to algae, aquatic invertebrates, and fish (see Brix et al. 2001, Schlenk et al. 2003, USEPA 2004). USEPA (2004) provides the following discussion on the effects of sulfate on selenium toxicity:

"The toxicity of a number of metals (e.g., copper and cadmium) to aquatic organisms is related to the concentration of hardness in the water. The toxicity of these metals to many different aquatic species has been shown to decrease as the hardness concentration increases. A similar relationship also has been recognized between selenate and dissolved sulfate in freshwater (a similar relationship is not evident between selenite and sulfate or between either form of selenium and hardness). The studies reviewed in this document indicate that, as the concentration of sulfate increases, the acute toxicity of selenate is reduced (less toxic). Selenate acute toxicity tests conducted at different levels of dissolved sulfate are available with C. dubia, D. magna, H. azteca, G. pseudolimnaeus*, chinook salmon and fathead minnows (Table 1a* [of USEPA 2004]*). These data indicate that, in general, selenate is more toxic to these species in low sulfate water than in higher sulfate water."*

Since sulfate levels should be higher in brackish and marine waters than in freshwaters, screening values based on freshwater selenium toxicity studies are likely to be protective of potential impacts to fishes in estuarine and marine environments. There are several possible reasons why sulfate may reduce the toxicity of selenate, including 1) the ability of sulfate, calcium, or magnesium to prevent the interaction of selenomethionine with critical subcellular proteins by maintaining the cellular redox potential (Schlenk et al. 2003) and 2) direct competition at the cell uptake site as selenate and sulfate are structurally similar group VI oxyanions of the form XO_4; experiments with bacteria indicate that selenate and sulfate have a common membrane carrier and that active transport by this carrier is the only means by which selenate may enter a cell (Brix et al. 2001).

Potential screening values for the North Bay

The objective of this review of the selenium toxicity information available for fishes is to provide a technical basis for the selection of an appropriate selenium fish tissue screening value for in the North Bay. Since there are limited selenium toxicity data available for fish of concern for the North Bay (e.g., no suitable selenium toxicity data for delta smelt, halibut, jacksmelt, striped bass, Tidewater goby, or white croaker), it is not possible to develop screening values that take into account the effects of selenium on all fish species of potential concern in the North Bay.

The SMCVs for species such as Chinook salmon, rainbow trout, Sacramento splittail, and white sturgeon indicate that these species are less sensitive to selenium than bluegill and channel catfish (Table 3-4), which appear to be fairly sensitive to selenium. Thus, using a screening value for the North Bay based on either bluegill (e.g., SMCV of 5.6 mg/kg-dw whole body) or channel catfish (e.g., SMCV of 3.0 mg/kg-dw whole body) should be protective of most (if not all) fish species, while not setting an unrealistically low threshold for potential effects.

The single study that evaluated the effects of selenium in both brackish and freshwater (Hamilton et al. 1990), indicated that Chinook salmon were more than twice as sensitive to selenium in freshwater as in brackish water. However, this study examined the effects of selenium on larvae in fresh water and juveniles in brackish water. Therefore, the greater sensitivity observed in freshwater with larvae could also be due to life history differences in selenium sensitivity.

Table 3-4 also presents SMCVs for selenium in liver and muscle. Liver concentrations respond rapidly to changes in the environment and may be useful in detecting short term selenium fluctuations. Muscle concentrations may be non-destructively measured in relatively large fishes by collecting and analyzing muscle plugs. This may be desirable as it reduces the impact of selenium monitoring on fish populations, which is especially important for special status species. However, it should be noted that the regressions used here to convert selenium concentrations measured in one tissue type to another tissue type are not specific to the species and populations considered here, with the exception of white sturgeon (see Appendix A). Therefore, using screening values that were calculated using the regressions from USEPA (2004) incorporates some uncertainty.

Beckon (2007)

In contrast to the approach used here (i.e., calculating effect thresholds and SMCVs), Beckon (2007) derived an EC10 and an EC20 (of 1.84 and 2.5 mg/kg-dw, respectively) for the survival of salmon fry by performing a regression on the treatment means in Hamilton (et al. 1990) (see above). However, it should be noted that performing a regression on the treatment means artificially reduces the variation. Further, the shape of the relationship may change when using the raw data. Thus, the EC10 and EC20 derived by Beckon (2007) should be viewed as approximations. It is also unknown whether the EC10 and EC20 are significantly different from each other.

Nutritional requirements

Selenium is a micro-nutrient (see Section 1). When animals do not have enough selenium in their diet, selenium deficiency results (NRC 1993, 1994). For fish, nutrient requirements are given in terms of the concentration in their feed, not in fish tissue, as is being used here to derive screening values. Nonetheless, the nutritional requirements of two species of fish have been established and are provided here for reference: 0.25 mg/kg for channel catfish and 0.3 mg/kg for rainbow trout (NRC 1993). These concentrations are given in an "as fed basis." Fish feed is usually in the form of dry pellets and probably has a moisture content of approximately 10%, meaning the dietary requirements listed above in mg/kg-as fed do not differ substantially from mg/kg-dw.

Additional considerations

Issues to be considered in the collection and interpretation of samples include:

- tissue concentrations in fishes may vary with the time of year sampled
- tissue concentrations may vary by geographic location
- if sampling dietary items of birds, selenium concentrations may also differ among their prey items
- number of samples to collect
- the frequency of collections
- whether samples should be composited
- how to compare measured concentrations to screening values

These issues are discussed in greater detail in Section 6.

The studies used in the determination of the screening value presented above, and all screening values obtained from the literature, were not derived using the populations of fish that occur in the North Bay nor using the environmental conditions in the North Bay. This means that there is some uncertainty as to whether any screening criteria are actually applicable. Similarly, McDonald and Chapman (2007), recommend that screening values only be used to determine if further evaluation is necessary. They recommend that if fish tissue concentrations exceed an appropriate screening value that 1) reproductive toxicity testing be conducted for the species of concern and that if reproductive toxicity testing indicates that the species has been negatively affected by selenium that 2) fish population surveys be conducted. McDonald and Chapman (2007) provide recommendations on how both reproductive toxicity testing and fish population surveys can be integrated into a selenium monitoring program.

Future research and data gaps

Several data gaps and areas where further research may be beneficial to the Water Board were identified during the evaluation of selenium toxicity to fish in the North Bay, as follows:

1. Although it is believed that selenium should be less toxic to fish in marine and estuarine systems than freshwater systems, the data to show this are lacking. Toxicity experiment performed with representative fish species in using multiple salinities over the range that occurs in the North Bay would provide valuable information.
2. Toxicity studies have not been performed on most of the species of concern (which includes endangered species) in the North Bay. Thus, this report has assumed that toxicity studies on bluegill, rainbow trout, chinook salmon, channel catfish, and

fathead minnows are representative of the species of concern. The species of concern may be more or less sensitive.

3. Channel catfish appear to be one of the most sensitive species. However, only two studies were available. If the Water Board believes that channel catfish are representative of the fish in the North Bay, additional toxicity studies using channel catfish would be warranted.

4. The existing toxicity data suggests that younger life history stages are more sensitive to selenium, but most selenium toxicity studies have focused on only a single life history stage. Thus, it is possible that some species may be more sensitive to selenium than shown here.

5. Species-specific regressions to convert selenium concentrations from one tissue type to another are lacking for all of the fish species evaluated here, except white sturgeon. Therefore, the regressions from USEPA (2004) for bluegill were used for most species. Since the applicability of these regressions to other species is unknown, screening values derived using the bluegill tissue relationships should be viewed as approximations and may need to be revisited as new data becomes available. Further, if the Water Board decides to monitor fish tissue concentrations by collecting muscle plugs, it would be beneficial to develop species-specific regressions.

6. A detailed re-analysis of the published toxicity data using, for example, biphasic regressions (Beckon 2007, Beckon et al. 2008) would provide a greater level of confidence in estimates of the threshold for toxic effects of selenium vs. a LOAEL, NOAEL, or effect threshold. However, if only the treatment means are available for the analyses, it should be remembered that this type of analysis will mask the true variability in the data.

3.4 CONCLUSIONS

Exposure to elevated levels of selenium can cause adverse effects in fish. Two of the species that occur in the North Bay that have been tested (i.e., Sacramento splittail and white sturgeon) appear to be less sensitive than other freshwater fish. While it has generally been believed that bluegill are one of the more sensitive fish species, the two studies available on channel catfish indicates that they may be more sensitive than bluegill. However, one of the most important concerns for selenium toxicity to fish remains an open question at the end of this review: is selenium less toxic to fish in marine and brackish waters than freshwater? The available data supports the assertion that selenium should be less toxic to fish in marine and brackish waters, but it is not possible to quantify the difference at this point.

Numerous selenium screening values protective of fish have been proposed. For whole body fish selenium concentrations, the recommended screening values are usually in the range of 2-4 mg/kg-dw (Table 3-5). Additionally, it has been proposed that separate screening values be used for cold water fish vs. warm water fish, although recent reviews have not found support this distinction (e.g., Hamilton 2003, Chapman 2007).

Based upon the data presented here, there are several options for selenium fish tissue screening values. Those options are as follows (all in units of mg/kg-dw whole body):

1. The lowest SMCV: 3.0 (Table 3-4)
2. The salmon EC20 from Beckon (2007) of 2.5
3. Lowest concentration at which adverse effects were reported (Table 3-3)
   a. LOAEL of 2.3
4. The lowest LOAELs for fish in the North Bay (Table 3-3)
   a. 12.9 for Sacramento splittail
   b. 12.3 for white sturgeon
5. The lowest effect thresholds for fish in the North Bay (Tables 3-3, 3-4)
   a.10.8 for Sacramento splittail
   b.6.2 for white sturgeon
6. The lowest SMCVs for fish in the North Bay (Table 3-4)
   a.10.8 for Sacramento splittail
   b.10.6 for white sturgeon
7. The white sturgeon EC10 calculated by Beckon (2008) from the data in Linville (2006) of 11.9

The choice of using an SMCV, LOAEL, or effect threshold is dependent upon both the level of risk and uncertainty the Water Board wishes to accept. This issue was discussed in more detail in the Introduction. It is worth noting that of these values, only the LOAELs represent concentration at which effects have been demonstrated. However, since only a few concentrations have been tested, effects may actually occur at lower concentrations.

4 Avian toxicity review

The waters of the North Bay supports a diverse avian biota. Some birds are residents in the Bay all year round, while many others rely on the North Bay as part of their annual migrations. The birds supported include both game birds as well as and threatened and endangered species. The North Bay supports the following threatened and endangered bird species (USFWS 2007):

- American bittern (*Botaurus lentiginosus*)
- American peregrine falcon (*Falco peregrinus anatum*)
- American white pelican (*Pelecanus erythrorhynchos*)
- bald eagle (*Haliaeetus leucocephalus*)
- black tern (*Chlidonias niger*)
- California black rail (*Laterallus jamaicensis coturniculus*)
- California brown pelican (*Pelecanus occidentalis californicus*)
- California clapper rail (*Rallus longirostris obsoletus*)
- California least tern (*Sterna antillarum browni*)
- double-crested cormorant (*Phalacrocorax auritus*)
- marbled murrelet (*Brachyramphus marmoratus*)
- mountain plover (*Charadrius montanus*)
- northern harrier (*Circus cyaneus*)
- osprey (*Pandion haliaetus*)
- prairie falcon (*Falco mexicanus*)
- tricolored blackbird (*Agelaius tricolor*)
- western least bittern (*Ixobrychus exilis hesperis*)
- western snowy plover (*Charadrius alexandrinus*)
- white-faced ibis (*Plegadis chihi*)
- white-tailed kite (*Elanus leucurus*)

The North Bay supports a number of birds that are not classified as threatened and endangered but are covered under the Migratory Bird Treaty Act (MBTA), including (USFWS 2007):

- Aleutian Canada goose (*Branta canadensis leucopareia*)
- black scoter (*Melanitta nigra*)
- Caspian tern (*Sterna caspia*)
- greater scaup (*Aythya marila*)
- lesser scaup (*Aythya affinis*)
- surf scoter (*Melanitta perspicillata*)
- white-winged scoter (*Melanitta fusca*)

Lastly, the North Bay supports several species of water fowl that are of recreational interest. While the California Department of Fish and Game (DFG) does not provide any information on which species are harvested in the North Bay, they do provide limits (i.e., "bag limits") on how many can be harvested at one time (DFG (2007). These limits, which indicate which water fowl species are of recreational interest, are provided below:

- canvasback (*Aythya valisineria*): 1
- American coot (*Fulica americana*): 25
- Ross's goose (*Chen rossii*): 4
- snow goose (*Chen caerulescens*): 4
- white-fronted goose (*Anser albifrons*): 4
- cackling goose (*Branta hutchinsii*): 6
- Aleutian goose (*Branta canadensis leucopareia*): 6
- Canada goose (*Branta canadensis*): 4
- mallard (*Anas platyrhynchos*): 7 but not more than 2 hens
- common moorhen (*Gallinula chloropus*): 25
- pintail (*Anas acuta*): 1
- redhead (*Aythya americana*): 2

- scaup (*Athya affinis and Athya marila*): 3
- merganser (*Mergus spp.* and *Lophodytes cucullatus*): 7

The USFWS (2007) evaluated the bird species found in the North Bay that are covered under the MBTA as well as the threatened and endangered species and determined that the following species were most at risk from potential exposures to selenium:

- bald eagle (*Haliaeetus leucocephalus*)
- black scoter (*Melanitta nigra*)
- California clapper rail (*Rallus longirostris obsoletus*)
- greater scaup (*Aythya marila*)
- lesser scaup (*Aythya affinis*)
- surf scoter (*Melanitta perspicillata*)
- white-winged scoter (*Melanitta fusca*)

Among birds, exposures to elevated levels of selenium can induce gross abnormalities during development, egg inviability, chick mortality (Adams et al. 2003), reduced weight gain, reduced adult condition, and reduced adult survival (NRC 2005, Ohlendorf 2003). When the selenium concentrations are high enough in a large enough area, these effects can lead to population declines for the most affected species.

The step in the effort to determine whether birds may be adversely impacted by selenium in the North Bay is to evaluate the toxicity of selenium to the marine mammals. Next, potential selenium screening values for the species in the North Bay are proposed. Lastly, the screening values are compared to nutritional requirements.

4.1 METHODS

Selenium toxicity data for the bird species that the USFWS (2007) has identified as being most at risk from selenium in the North Bay (see list above) would be the most relevant for the Water Board's development of a TMDL. However, selenium toxicity studies have not been performed for any of the species identified by the USFWS (2007) as being most at risk from selenium in the North Bay. Therefore, in this section of the report, we review the available avian selenium toxicity literature.

For birds, selenium studies have generally focused on 1) determining a toxic concentration in the diet, 2) determining a toxic concentration in eggs, or 3) improving bird "performance" on a poultry farm. We reviewed papers published in the scientific literature for all three types of data. Studies were excluded from the consideration for the selection of a screening value (see Table 4-1) if they:

A. did not provide a LOAEL

B. were not chronic

C. exposed birds to multiple contaminants simultaneously

D. exposed birds to multiple treatments simultaneously

E. field studies

Using the filtering criteria above, only studies where the reported effects can unambiguously be attributed to selenium were reviewed. Some of the poultry performance studies were not performed using dietary concentrations high enough to cause toxic effects. These studies will be used to set a lower bound on screening values. Most of the studies describing the early laboratory work on the effects of selenium on poultry species are no longer readily accessible. When the original studies are not readily available, the reviews that summarize the results of the original studies are cited.

The reported effects from each study were grouped into one of two categories: major and minor effects. Major effects are those that have the potential to impact birds at the organism and/or population level (e.g., increased mortality, reduced fecundity, reduced growth, etc.). Minor effects are those that are measurable, but are unlikely to result in population level effects and/or the long-term impacts on individual bird performance was unclear (e.g., a change in an enzyme concentration). Where both major and minor effects were observed at the same concentration, the effects was assumed to be major. Only major effects were considered in development of screening values.

All studies that were reviewed are listed in Table 4-1. Studies that were determined to be unsuitable are noted in Table 4-1 using the designations from the list above. The 21 suitable studies selected are presented in Table 4-2.

Table 4-1
Selenium toxicity studies evaluated in this review. Studies that were excluded from review are indicated in the table (see text above for an explanation of the criteria).

Study	Reference	Evaluation criteria failed	Study	Reference	Evaluation criteria failed
1	Albers et al. (1996)		24	Latshaw et al. (2004)	
2	Anteau et al. (2007)	A,C,E	25	Lowry and Baker (1989)	
3	Biswas et al. (2006)	A	26	Ort and Latshaw (1978)	
4	Elzubier and Davis (1988)		27	O'Toole and Raisbeck (1997)	
5	Fairbrother et al. (1994)	C,E	28	Pappas et al. (2005)	A
6	Franson et al. (2007)		29	Paton et al. (2002)	A
7	Green and Albers (1997)		30	Payne et al. (2005)	A
8	Harding (2007)	E	31	Ratti et al. (2006)	A,E
9	Heinz and Hoffman (1996)		32	Rattner et al (2000)	A,C,E
10	Heinz and Hoffman (1998)		33	Ryu et al. (2005)	A
11	Heinz et al. (1987)		34	Sahin and Kucuk (2001)	A
12	Heinz et al. (1988)		35	Santolo et al. (1999)	A
13	Heinz et al. (1989)		36	Stanley et al. (1994)	
14	Heinz et al. (1996)		37	Stanley et al. (1996)	
15	Hoffman and Heinz (1998)		38	Stoewsand et al. (1978a)	A,C
16	Hoffman et al. (1991)		39	Stoewsand et al. (1978b)	A,C
17	Hoffman et al. (1992a)		40	Surai et al. (2006)	A
18	Hoffman et al. (1992b)		41	Takekawa et al. (2002)	A,C,E
19	Hoffman et al. (1996)		42	Wayland et al. (2002)	A,C,E
20	Hoffman et al. (1998)	C,E	43	Wiemeyer and Hoffman (1996)	
21	Hoffman et al. (2002)	C,E	44	Wilson et al. (2007)	A,C,D,E
22	Jensen (1975)		45	Yamamoto and Santolo (2000)	A
23	Kinder et al. (1995)	D	46	Yamamoto et al. (1998)	A

To be consistent with the approach used for developing screening values for fish, effect thresholds were calculated as the geometric mean of the NOAEL and LOAEL reported for the same effect in an individual study. Additionally, species mean chronic values (SMCVs) were calculated as the geometric mean of the effect thresholds (USEPA 1985). Studies in which a zero concentration was reported in the control were assumed to have used 0.2 mg/kg-dw, as this is the dietary requirement for birds (NRC 1984). Studies that did not report a NOAEL were excluded from the calculation of an effect threshold and the SMCV. Note that excluding studies in which a NOAEL was not reported from the calculation of the SMCV results in lower SMCVs.

Conversions from dry weight to wet weight are detailed in Appendix A.

4.2 RESULTS

Toxicity studies

The toxicity studies from Table 4-1 that were included in the evaluations below included the following bird species:

- Chickens
- Eiders
- Kestrels
- Pheasants

- Mallards
- Screech owls

In contrast to the studies on fish, where whole body selenium concentrations are usually measured, the toxicity studies performed on birds have usually measured the selenium content of the feed given to the bird. Therefore, the screening values derived here are for dietary selenium concentrations.

Chickens

Four studies were identified for domestic chickens (Table 4-2). Only one study (Ort and Latshaw 1978) examined the effect of selenium on the most sensitive endpoint; i.e., egg hatchability. It is also worth noting that Ort and Latshaw (1978) review two earlier studies not available to us that show 1) “egg weight, fertility and hatchability were all significantly decreased by 8 ppm [dietary] selenium,” and 2) “Embryonic development was adversely affected by 10 ppm dietary selenium but not by 5 ppm.”

Table 4-2
Summary of dietary selenium toxicity studies evaluated in this review.

Bird	Study	Life stage	Dietary form	(mg/kg-dw)			Effect		Endpoint
				NOAEL	LOAEL	Effect threshold	Major	Minor	
Chicken	4	Chick	selenite	0.15	10	1.2	X		BW
Chicken	22	Chick	selenite	0.9	4.3	2.0	X		BW
Chicken	22	Chick	selenite	4.3	13.5	7.6	X		BW, S
Chicken	25	Chick	selenite; SeMet	0.2	15	1.7	X		BW
Chicken	26	Adult	selenite	3	5	3.9	X		R
Common eider	6	Adult	SeMet	20.6	57.7	34.5	X		BW
Mallard	1	Adult	SeMet	22.3	44.7	31.6	X		C
Mallard	7	Adult	SeMet	11.2	22.3	15.8	X		S
Mallard	9	Adult	SeMet	0.6	10.6	2.6	X		R
Mallard	10	Adult	SeMet	0.4	9.8	2.1	X		R
Mallard	11	Adult	selenite	10.9	27.3	17.3	X		BW, R
Mallard	11	Adult	SeMet	0.2	10.9	1.5	X		R
Mallard	12	Duckling	selenite	10.9	21.6	15.3	X		BW
Mallard	12	Duckling	SeMet	10.9	21.6	15.3	X		BW
Mallard	13	Adult	SeMet	4.6	9.0	6.4	X		R
Mallard	14	Duckling	SeMet	17.0	33.7	24.0	X		BW
Mallard	15	Adult	SeMet	0.4	11.6	2.3		X	E
Mallard	16	Duckling	SeMet; low protein	0.2	16.9	1.9	X		BW
Mallard	16	Duckling	SeMet	16.9	66.9	33.6	X		BW, S
Mallard	17	Duckling	SeMet	0.2	16.9	1.9	X		BW
Mallard	18	Duckling	SeMet	0.2	16.5	1.9		X	E
Mallard	18	Duckling	SeMet	16.5	65.4	32.9	X		BW
Mallard	18	Duckling	SeMet; low protein	16.5	65.4	32.9	X		S
Mallard	18	Duckling	SeMet; low protein	0.2	16.5	1.9		X	E
Mallard	18	Duckling	SeMet; high protein	0.2	16.5	1.9	X		BW
Mallard	19	Duckling	SeMet, Se yeast, Se in wheat	0.2	16.9	1.9		X	E
Mallard	27	Flightling	SeMet	13.8	33.8	21.6	X		BW, S
Mallard	36	Adult	SeMet	0.37	6.5	1.6	X		R
Mallard	37	Adult	SeMet	3.9	7.8	5.5	X		BW, R
Pheasant[1]	24	Adult	unknown	0.4	9.3	1.9	X		R, S
Screech owl	43	adult	SeMet	8.8	30	16.2	X		BW, R
Screech owl	43	adult	SeMet	0.3	8.8	1.6		X	E

Notes: 1 - Although the concentration at the NOAEL was not measured, the lead investigator on this study said that he believes it was probably 0.4 mg/kg.
BW = Body Weight
C = Condition
D = Deformities
E = Enzymes
FG = Feed:Gain ratio
L = Length
R= Reproduction
S = Survival
SA = Swimming Activity

In addition to the studies summarized in Table 4-2, the NRC (1980, 1994, 2005) provide summaries of additional dietary selenium toxicity studies on domestic chickens that were not available to the authors of this report. These data are presented in Tables 4-3 through 4-5, respectively.

Table 4-3
Summary of additional selenium toxicity studies from the NRC (1980).

Age	Chemical Form	Concentration (mg/kg)		Effect	Reference
		NOAEL	LOAEL		
Adult	Se in corn, barely, wheat	2.5	5	increased chick mortality	Moxon (1937)
Adult	Na_2SeO_3	-	6.5	deformed embryos	Moxon (1937)
1 day	Na_2SeO_3	4	8	decreased weight gain	Moxon (1937)
1 day	selenous acid	2	8	increased chick mortality	Thapar et al (1969)
1 day	SeO_2	-	5	increased mortality	Hill (1979)
Adult	Na_2SeO_3	-	8	embryo necrosis	Gruenwald (1958)

Table 4-4
Summary of additional selenium toxicity studies from the NRC (1994).

Age	Chemical Form	Concentration (mg/kg)	Effect	Reference
Immature	Na_2SeO_3 + Se in wheat	10	Reduced growth	Carlson and Leitis (1957)
Laying hen	Se in wheat	10	Reduced hatchability	Moxon and Wilson (1944)

Table 4-5
Summary of additional selenium toxicity studies from the NRC (2005).

Age	Chemical Form	Concentration (mg/kg)		Effect	Reference
		NOAEL	LOAEL		
Adult	selenite	5	10	reduced growth	Jensen and Chang (1976)
1 day	selenite	1	5	reduced growth	Jensen (1986)

For all of the studies on chickens presented above, it is unclear whether the dietary concentrations are provided as ww or dw. It is likely that the dietary concentrations are ww but that the %moisture is relatively low (e.g., 10%) as chicken feed is usually in the form of dry pellets. So, there is some uncertainty about the exact concentration of selenium in the studies above on chickens, but it is likely to be a small effect; e.g., 5 mg/kg-ww with a %moisture of 10% is equivalent to 5.6 mg/kg-dw.

Eiders
A single suitable study was identified (Franson et al. 2007). That study provided both a NOEAL and LOAEL for weight loss (i.e., a major effect) in diet, but did not examine reproductive success (Table 4-2).

Kestrels
Three studies have been conducted on the toxicity of dietary selenium to kestrels (Santolo et al. 1999, Yamamoto et al. 1998, Yamamoto and Santolo 2000). The first two studies (i.e., Santolo et al. 1999, Yamamoto et al. 1998) did not show an effect at up to 12 mg/kg-dw. In the third study (Yamamoto and Santolo 2000), the authors state that kestrels fed 12 mg/kg-dw selenium had a lower "normalized body fat" than the kestrels in the other treatments.

However, the difference was not significant (i.e., $p = 0.173$) and was, therefore, excluded from Table 4-2 and consideration in the development of a screening value.

Mallards

Mallards are the bird species for which the most information is available. Fourteen suitable studies were identified on mallards. The studies examined effects on adults, ducklings, and reproductive success (Table 4-2).

Pheasants

A single suitable study was identified (Latshaw et al. 2004). That study provided a NOAEL and LOAEL based on mortality and reduced egg laying (Table 4-2).

Screech owls

A single suitable study was identified (Wiemeyer and Hoffman 1996). That study provided both a NOAEL and LOAEL for selenium in the diet and eggs based on weight loss and reduced reproductive success. A NOAEL and LOAEL was also provided for minor effects; i.e., changes in enzyme concentrations (Table 4-2).

4.3 IMPROVED PERFORMANCE STUDIES

Among birds that are raised on poultry farms, there has been extensive research in an attempt to balance the beneficial effects of selenium supplementation against the toxicity of selenium. These studies generally provide NOAELs only and do not necessarily examine the effects of selenium on reproductive success. The available studies are summarized in Table 4-6 below.

Table 4-6
Studies evaluating improved performance in poultry from selenium supplementation.

Bird	Study	Life stage	Dietary form	NOAEL (mg/kg)	dw/ww		Effects			
							Survival	Weight	Egg output	Reproductive success
Chicken	24	Adult	selenite	2.6	?				X	
Chicken	24	Adult	Se-yeast	2.9	?				X	
Chicken	27	1 day	selenite	8.17	dw			X		
Japanese quail	3	1 day	selenite	1.2	?		X	X		
Japanese quail	28	10 day	selenite	0.2	dw			X		

The most sensitive endpoint for selenium effects in birds is reproductive success (i.e., egg hatchability and chick survival). However, since none of the performance studies examined the potential effects of selenium on reproductive success, these studies should not be used to put a lower bound on the acceptable level of dietary selenium for birds.

4.4 DISCUSSION

More than 40 selenium toxicity studies were reviewed in an effort to determine dietary selenium screening values for species representative of birds in the North Bay (Table 4-1). Based on this review, species which are appropriate for use in developing screening criteria for the North Bay, and for which usable selenium toxicity data are available, include:

chickens (*Gallus gallus*), common eiders (*Somateria mollissima*), mallards (*Anas platyrhynchos*), pheasants (*Phasianus colchicus*), and screech owls (*Otus asio*).

The data presented above (Tables 4-2 through 4-5; regardless of selenium type) were grouped into effects on immature birds (i.e., chicks and ducklings), adults, and reproductive success (i.e., egg hatchability, chick survival, egg fertility, etc.) and are presented in Table 4-7 below.

Table 4-7
Summary of NOAELs and LOAELs (mg/kg-dw) reported in this section grouped by whether the effects were measured in chicks/ducklings, adult birds, or in terms of reproductive success. Studies using all selenium species are shown here.

	Chick/Duckling			Adult			Reproductive success		
	NOAEL	**LOAEL**	**Effect Threshold**	**NOAEL**	**LOAEL**	**Effect Threshold**	**NOAEL**	**LOAEL**	**Effect Threshold**
Chicken	0.15	10	1.2	5	10	7.1	2.5	5	3.5
	0.2	15	1.7				3	5	3.9
	0.9	4.3	2.0				5	10	7.1
	1	5	2.2				-	6.5	-
	2	8	4.0				-	8	-
	4	8	5.7				-	8	-
	4.3	13.5	7.6				-	10	-
	-	5	-						
	-	10	-						
		SMCV	2.9		SMCV	7.1		SMCV	4.6
Eider				20.6	57.7	34.5			
Mallard	0.2	16.5	1.9	11.2	22.3	15.8	0.2	10.9	1.5
	0.2	16.9	1.9	13.8	33.8	21.6	0.37	6.5	1.6
	0.2	16.9	1.9	22.3	44.7	31.6	0.4	9.8	2.1
	10.9	21.6	15.3				0.6	10.6	2.6
	10.9	21.6	15.3				3.9	7.8	5.5
	16.5	65.4	32.9				4.6	9.0	6.4
	16.5	65.4	32.9				10.9	27.3	17.3
	16.9	66.9	33.6						
	17.0	33.7	24.0						
		SMCV	10.4		SMCV	22.1		SMCV	3.6
Pheasant							0.4	9.3	1.9
Screech owl							8.8	30	16.2

From the data presented in Table 4-7, it would appear that immature chickens are more sensitive to selenium than immature mallards, although there was some variation among chicken strains (see Table 4-2). The available studies on reproductive success appear to indicate that mallards are a little more sensitive than chickens but that screech owls are less sensitive than either chickens or mallards. For pheasants, the small amount of available data indicates that reproductive success is affected at relatively low concentrations.

State of Utah

The State of Utah is also deriving dietary selenium screening values protective of birds feeding at the Great Salt Lake (Ohlendorf 2007, provided as Appendix B). To develop a selenium screening value for the Great Salt Lake, the Great Salt Lake Science Panel compiled the concentration of selenomethionine in feed, total selenium in eggs, and mean hatching success per treatment from 6 studies on mallards (i.e., Heinz et al. 1987, 1989, Heinz and Hoffman 1996, 1998, Stanley et al. 1994, 1996; all of these studies are included in Tables 4-1, 4-2, and 4-7) and examined the data using both a logistic regression and a hockey-stick regression. The regressions were used to derive concentrations at which 10% of the population would be affected (EC10) for both dietary and egg selenium concentrations. The results for mean hatching success are as follows (mg/kg-dw):

- Dietary selenium
  - Logistic regression: 4.9
  - Hockey stick-regression: 4.4
- Egg selenium
  - Logistic regression: 12.5
  - Hockey stick-regression: 11.5

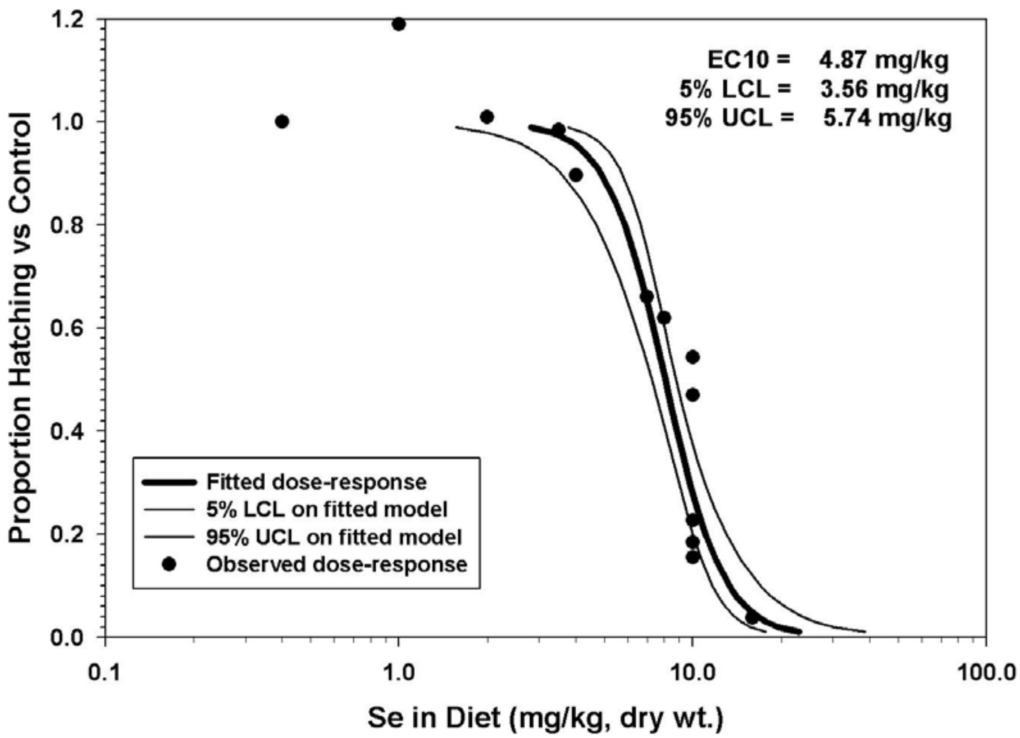

Figure 4-1 **Logistic regression of mallard dietary selenium concentration vs. proportion of eggs hatching. From Ohlendorf (2007).**

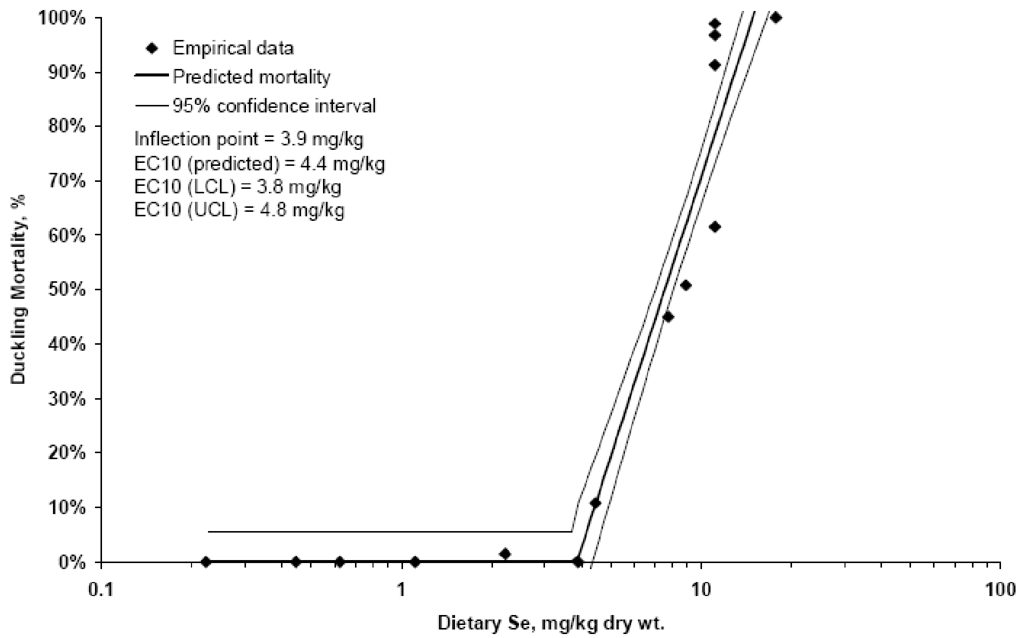

Figure 4-2 **Hockey stick regression of mallard dietary selenium concentration vs. proportion of eggs hatching. From Ohlendorf (2007).**

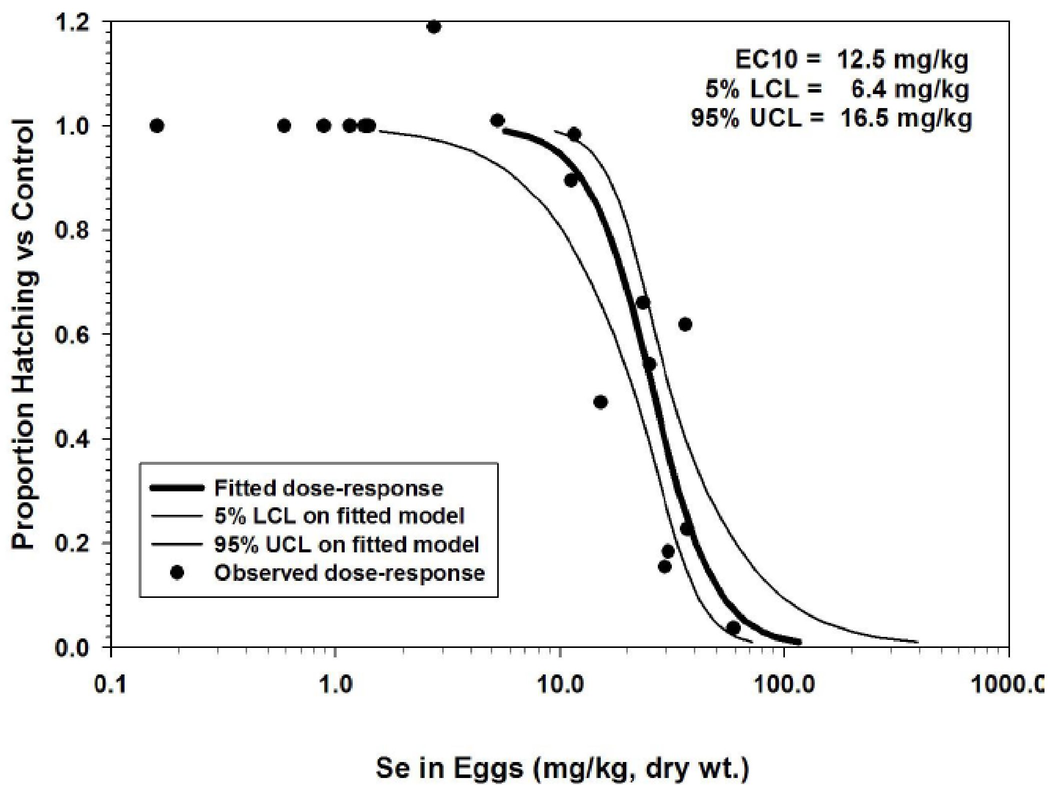

Figure 4-3 **Logistic regression of mallard egg selenium concentration vs. proportion of eggs hatching. From Ohlendorf (2007).**

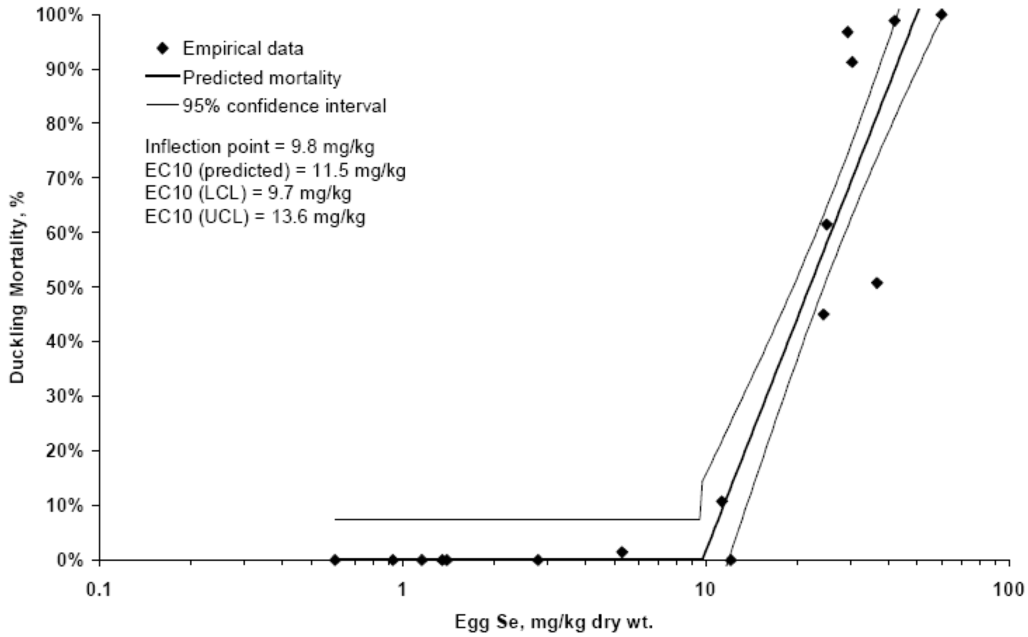

Figure 4-4 Hockey stick regression of mallard egg selenium concentration vs. proportion of eggs hatching. From Ohlendorf (2007).

The logistic regressions were previously reported in Ohlendorf (2003) and the hockey-stick regression for egg selenium was previously reported in Adams et al. (2003). The paper by Adams et al. (2003) is the third paper published by the same group of authors on this topic; i.e., Fairbrother et al. (1999, 2000) used the data from fewer studies (i.e., Heinz et al. 1989 and Stanley et al. 1994) to derive an EC10 for eggs of 16 mg/kg-dw.

As discussed above for the regression approach used by Beckon (2007) to analyze fish data, the State of Utah (Ohlendorf 2007) performed regressions on the treatment means, instead of the raw data from each individual, which artificially reduces the variation; i.e., the confidence limits on the regression lines in Figures 4-1 through 4-4 may be much larger. Further, the shape of the relationship may change when using the raw data. Thus, the EC10 and EC20 presented in Figures 4-1 through 4-4 should be viewed as approximations. Lastly, the data used in the logistic regressions (Ohlendorf 2003) was also normalized against the controls. This may have obscured a biphasic relationship (see Beckon et al. (2008)).

There are a few minor problems with the data used by the State of Utah (Ohlendorf 2007) that should be kept in mind. First, the concentration of selenomethionine used in the logistic regressions was assumed by the State of Utah (see Ohlendorf 2003) to be dry weight (Ohlendorf 2007) but the concentrations were actually presented as wet weight. The State of Utah (Ohlendorf 2007) acknowledged this by stating that the values derived in the logistic regressions for dietary concentrations should be increased by 11 percent (i.e., to 5.4 mg/kg-dw). Further, the State of Utah (see Ohlendorf 2003) used the "nominal" selenium concentrations added to feed in their analyses and not the measured concentrations. This adds an additional layer of uncertainty to the thresholds reported by the State of Utah (Ohlendorf 2007) as the actual concentration can differ from the nominal concentration either positively or negatively, depending upon the individual treatment.

Other selenium screening values

The National Irrigation Water Quality Control Program (NIWQP) recommends a lower screening value for selenium in bird eggs than the State of Utah; i.e., 6 mg/kg-dw (NIWQP 1998). The USGS (Seiler et al. 2003) used logistic regressions to derive threshold concentrations for selenium induced teratogenicity (a less sensitive endpoint than hatching success) in bird eggs collected from several selenium-impacted field sites across California (Table 4-8). The NIWQP also recommends a screening value for dietary selenium of 3 mg/kg-dw. Presser and Luoma (2006) also reviewed the toxicity literature and concluded that dietary "concern levels range from 2 to 7" mg/kg-dw for birds.

Table 4-8
Selenium screening values for teratogenesis in bird eggs (mg/kg-dw) (Seiler et al. 2003).

Bird	EC01	EC10
Ducks	15	23
Black-necked stilts	14	37
American avocets	41	74

Allometric adjustment for dietary concentrations

The species of concern in the North Bay identified by USFWS (2007) have not been examined in selenium toxicity studies. One approach for applying screening values developed for one species to another species is to allometrically adjust the screening values (Sample and Arenal 1999). This adjusts for differences in body weight, metabolism, pharmacokinetics, and sensitivity to provide the best available estimates of species-specific toxicity. The equations use to perform the allometric adjustments, and parameter values, are provided in Appendix C.

Allometric adjustments were applied to predict dietary selenium screening values for the species of concern in the North Bay identified by USFWS (2007) from three example dietary toxicity values protective of reproductive success:

1. the EC10 for mallards fed selenomethionine derived using a hockey-stick regression by Adams et al. (2003) and used by the Great Salt Lake Science Committee (Ohlendorf 2007);
2. the effect threshold (Table 4-2) derived from the study by Ort and Latshaw (1978) for chickens, and;
3. the effect threshold (Table 4-2) derived from the study by Wiedemeyer and Hoffman (1996) for screech owls.

These studies were chosen as the most representative of the available toxicity data although other toxicity values may also be used (e.g., the SMCV for chicken reproductive studies). However, since reproductive success is the most sensitive endpoint in birds, it is recommended that the allometrically adjust dietary threshold all be based on that endpoint. The allometrically adjusted screening values are presented below in Table 4-9.

Table 4-9
Allometrically adjusted dietary selenium screening values.

Bird species	Dietary Screening Value (mg/kg-dw) Allometrically Adjusted from		
	Mallard[a]	Chicken[b]	Screech Owl[c]
Bald eagle	9.7	4.0	104.3
California clapper rail	2.2	0.9	-
Greater scaup	3.9	1.6	-
Lesser scaup	3.2	1.3	-
White-winged scoter	5.6	2.3	-
Surf scoter	4.1	1.7	-
Black scoter	3.9	1.6	-

Notes:
a - EC10 for reduced hatching success from Adams (2003) and Ohlendorf (2007) of 4.4 mg/kg-dw
b - effect threshold for reduced hatching success of 3.9 mg/kg-dw from Ort and Latshaw (1978) (Table 4-2)
c - effect threshold for reduced hatching success of 17.3 mg/kg-dw from Wiemeyer and Hoffman (1996) (Table 4-2)

Note that allometric adjustments are used to adjust dietary screening values (Sample and Arenal 1999) and are not used to adjust screening values for selenium in eggs. Further, there is some uncertainty associated with the use of allometric scaling as the allometric scaling factor used here was developed for acute toxicity data its "…*applicability to chronic toxicity data is unknown. The modes of action for acute and chronic effects differ for many chemicals. As a consequence, it is likely that scaling factors based on chronic toxicity data will also differ from those based on acute toxicity data*" (Sample and Arenal 1999). It should also be noted that the allometric scaling factor used here is not specific to selenium, but a mean value for other contaminants.

Allowable limits in bird feed

The FDA has approved the use of sodium selenate, sodium selenite, and selenium yeast at up to 0.3 mg/kg for chickens, swine, turkeys, sheep, cattle, and ducks (21CFR573.920). The major (i.e., 60-80%) form of selenium in selenium yeast is selenomethionine (Capelo et al. 2004, McSheehy et al. 2005, Polatajko et al. 2005).

Nutritional requirements

Selenium is a micro-nutrient. When animals do not have enough selenium in their diet, selenium deficiency results; i.e., poor growth, muscular dystrophy, and chick mortality (Ullrey 1992, NRC 1994). Due to the economic importance of the poultry industry, the nutritional requirements to maintain an adequate growth rate and normal metabolism of some of the more widely raised species have been established by the NRC (1994; see Table 4-10 below).

Table 4-10
Selenium nutritional requirements for commercially reared birds (mg/kg-dw).

Species	Strain	Age (weeks)			
		0-2	2-4	4-6	6-
Chicken	Leghorn white egg-laying	0.17	0.17	0.17	0.17
Chicken	Leghorn brown egg-laying	0.16	0.16	0.16	0.11
Chicken	Broiler	0.17	0.17	0.17	0.17
Turkey	-	0.22	0.22	0.22	0.22
Duck	-	0.22	-	-	-
Quail	Japanese	0.22	0.22	0.22	0.22

Source: NRC (1994)

Maximum tolerable limits

The NRC (2005) defines the maximum tolerable level as the concentration "…of a mineral is the dietary level that, when fed for a defined period of time, will not impair animal health and performance." The NRC (2005) has set the following maximum tolerable levels for selenium in bird diets: a) 3 mg/kg for poultry and b) 2 mg/kg-dw for aquatic birds. Poultry includes chickens, turkeys, grouse, and pheasants, as well as ducks and geese (which are water birds). It should also be noted that the studies on kestrels did not produced adverse effects at up to 12 mg/kg-dw (Santolo et al. 1999, Yamamoto et al. 1998, Yamamoto and Santolo 2000).

Additional considerations

Issues to be considered in the collection and interpretation of samples include:

- tissue concentrations in fishes may vary with the time of year sampled
- tissue concentrations may vary by geographic location
- if sampling dietary items of birds, selenium concentrations may also differ among their prey items
- number of samples to collect
- the frequency of collections
- whether samples should be composited
- how to compare measured concentrations to screening values

These issues are discussed in greater detail in Section 6.

A point worthy of consideration is whether the type of food consumed affects the bioavailability and/or toxicity of selenium. The bird toxicity studies reviewed above would likely have used a feed that mainly consisted of cereal grains and oilseed meals (NRC 1994), whereas many of the bird species of concern in the North Bay are at least partially carnivorous. Among pigs, "*animal Se sources generally have a lower (<25%) absorption rate than Se from plant tissue. However, Se absorption is influenced by the type of animal product being fed. For example, the absorption of Se from bovine milk is high and equivalent to that of selenite, whereas Se in meat and bone meal or poultry by-products is ≤20%*" (Mahan 2001). This implies that the bioavailability of selenium is likely to be lower in the diets of the birds in the North Bay than in the diets used in the toxicity studies reviewed here. Thus, dietary screening levels for selenium may need to be adjusted upwards when they are based on studies where the test animal was fed mainly vegetable matter.

The toxicity values presented above were not derived using the populations and species of birds that occur in the North Bay nor using the environmental conditions in the North Bay. This means that there is some uncertainty as to whether any screening criteria are actually applicable. Similarly, McDonald and Chapman (2007) recommend that screening values only be used to determine if further evaluation is necessary. They recommend that if sample concentrations exceed an appropriate screening value that actual impacts on the species of concern be assessed. Since reproductive success is the most sensitive endpoint in birds, it may be relatively easy to determine whether reproductive success of aquatic birds is being adversely impacted in the North Bay. This would require that an egg monitoring program be designed and implemented.

Future research and data gaps

Several data gaps and areas where further research may be beneficial to the Water Board were identified during the evaluation of selenium toxicity to birds in the North Bay, as follows:

1. The logistic regressions developed by the State of Utah (Ohlendorf 2007) to derive a dietary effect threshold for selenium should be redone using the correct data.
2. Toxicity studies have not been performed on most of the species of concern in the North Bay (USFWS 2007). Thus, this report has assumed that toxicity studies on mallards, chickens, pheasants, and screech owls are representative of the species of concern. The species of concern may be more or less sensitive.
3. If toxicity testing is not performed for the species of concern in the North Bay, another approach may be to develop selenium-specific allometric scaling factors for organic selenium in avian diets.
4. Species-specific regressions to convert dietary selenium concentrations to egg concentrations are lacking. If the Water Board decides to use the protection of egg hatching as a numeric criterion, this will be an important data gap.
5. The selenium toxicity studies performed to date have used selenite, selenomethionine, and selenized yeast. The degree to which the dietary formulations used in the toxicity studies reflects the speciation of selenium speciation in the food of birds of concern in the North Bay is not very well known. Since the speciation of selenium affects its toxicity, this may be an important consideration.
6. Toxicity experiments are uneven in assaying the actual amount of selenium in the experimental feeds. Many studies only report the estimated concentration added. Thus, the reported amount of selenium provided to birds in selenium toxicity studies is subject to some error. The error can be either positive or negative, depending upon the individual treatment.
7. Many toxicity studies performed to date report their concentration data in wet weight and do not provide a %moisture. Although a default %moisture can be used to estimate dry weight concentrations, this adds an additional layer of uncertainty as the actual %moisture will vary from sample to sample, at the very least.
8. A detailed re-analysis of the published toxicity data using, for example, biphasic regressions (Beckon 2007, Beckon et al. 2008) would provide a greater level of confidence in estimates of the threshold for toxic effects of selenium vs. a LOAEL, NOAEL, or effect threshold. However, if only the treatment means are available for the analyses, it should be remembered that this type of analysis will mask the true variability in the data.

4.5 Conclusions

Exposure to elevated levels of selenium can cause adverse effects in birds. Raptors, such as screech owls and kestrels, appear to be less sensitive to dietary exposures to selenium than ducks and chickens. While it has generally been recognized that reproductive success is the most sensitive endpoint in mallards, a review of the toxicity data available for chickens indicates that the growth/survival of young is a more sensitive endpoint in chickens. Thus, it is an open question as to which endpoint will be more sensitive for the birds of concern in the North Bay.

Numerous selenium screening values protective of birds have been proposed. For dietary selenium, the recommended screening values are usually in the range of 3-5 mg/kg-dw. For bird eggs, the range of recommended screening values is considerably larger, ranging from 6 to 74 mg/kg-dw.

Based upon the data presented here, there are several options for selenium screening values protective of birds. Those options are as presented below (all in units of mg/kg-dw). All of the screening values are well above the dietary requirements for birds of approximately 0.2 mg/kg-dw (Table 4-10), with the exception of some of the allometrically adjusted dietary thresholds, which are close to the dietary requirements.

1. The lowest dietary SMCV of 2.9 (Table 4-7)
2. Lowest dietary concentration at which adverse effects were reported (Table 4-7)
    a. LOAEL of 4.3
3. The effect concentrations protective of reproductive success in mallards derived by the State of Utah (Ohlendorf 2007):
    a. Dietary selenium
        i. Logistic regression: 5.4
        ii. Hockey stick-regression: 4.4
    b. Egg selenium
        i. Logistic regression: 12.5
        ii. Hockey stick-regression: 11.5
4. Allometrically adjusted dietary thresholds for the species of concern in the North Bay (Table 4-9)
    a. Note that allometric adjustment predicts that smaller birds will have lower dietary thresholds, with the lowest being approximately 1 mg/kg-dw for California clapper rails

The choice of using an SMCV, LOAEL, effect threshold, an EC01, or an EC10 is dependent upon both the level of risk and uncertainty the Water Board wishes to accept. This issue was presented in more detail in the Introduction. It is worth noting that of these values, only the LOAEL represents concentrations at which effects have been demonstrated. However, since only a few concentrations have been tested, effects may actually occur at lower concentrations.

5 MARINE MAMMAL TOXICITY REVIEW

Although some whale and dolphin species may occasionally enter the waters of the North Bay, they are generally considered infrequent visitors. The three marine mammal species that are considered to be resident in the North Bay include (DFG 2007, USFWS 1992):

- River otter (*Lontra canadensis*)
- Harbor seal (*Phoca vitulina*)
- California sea lion (*Zalophus californianus*)

Radio telemetry data indicates that harbor seals and sea lions venture in and out of the North Bay (USFWS 1992). None of the three marine mammal species found in the North Bay are special status species.

Among mammals exposure to elevated levels of selenium can cause reduced growth, emaciation, hair loss, lesions, paralysis, reduced conception rates, reduced litter size, increased stillbirths, and mortality (Mahan 2001, NRC 1980, 2005, Ohlendorf 2003). When selenium concentrations are high enough in a large enough area, these effects can lead to population declines for the most affected species.

The step in the effort to determine whether marine mammals may be adversely impacted by selenium in the North Bay is to evaluate the toxicity of selenium to the marine mammals. Next, potential selenium screening values for the species in the North Bay are proposed. Lastly, the screening values are compared to nutritional requirements.

5.1 METHODS

No toxicity studies have been performed on the effects of selenium on any marine mammal. Among mammals, selenium toxicity studies have been performed on cows, dogs, goats, hamsters, mice, pigs, and rats. However, "*due to the reduction of selenite and selenate, and the formation of insoluble particles in the rumen, cattle and sheep have [a] lower absorption of selenium… than non-ruminant species*" (NRC 2005). Therefore, toxicity studies where selenium has been fed to cows, sheep, and goats are probably not representative of marine mammals. Herbivorous and semi-herbivorous rodents (e.g., hamsters, mice, and rats) have reducing conditions in their intestines (as opposed to the stomach of ruminants) and also engage in coprophagy. Therefore, dietary toxicity studies on hamsters, mice, and rats are not likely to be reflective of carnivores, including marine mammals. Thus, it was assumed that dietary toxicity studies on pigs and dogs are likely to be representative of marine mammals.

Dietary selenium toxicity studies using pigs have been reviewed by the NRC (1980, 2005) and Mahan (2001). Studies on dogs have also been reviewed by the NRC (1980). The studies included in those reviews are summarized below in the Results section.

5.2 RESULTS

As stated above, no selenium toxicity studies on marine mammals were found. However, it was assumed that dietary toxicity studies on pigs and dogs are representative of marine mammals as all three groups are carnivorous mammals. The studies on pigs and dogs reviewed by the NRC (1980, 2005) are provided in Tables 5-1 and 5-2 below, respectively.

Table 5-1
Dietary toxicity studies on pigs from NRC (1980, 2005).

Age	Chemical Form	Concentration (mg/kg) NOAEL	LOAEL	Effect	Reference
"growing"	Na_2SeO_3	4	8	reduced weight gain	Goehring et al. (1984)
"growing"	Na_2SeO_3	5	15	reduced weight gain	Mahan and Magee (1991)
8-10 wk	Na_2SeO_3 and SeMet	0.4	25	reduced weight gain, paralysis	Panter et al. (1996)
24.7 kg	Na_2SeO_3 or Se-yeast	5	10	reduced weight gain	Kim and Mahan (2001a)
Adult	Na_2SeO_3	4	8	offspring with reduced weight	Poulsen et al. (1989)
25 kg	Na_2SeO_3 or Se-yeast	3	7	lower number of live born	Kim and Mahan (2001b)
5 kg	Na_2SeO_3 and SeMet	10	20	reduced weight gain	Herigstad et al. (1973)
35 kg	Seleniferous corn	5	10	toxicosis	Schoening (1936)
15 kg	Na_2SeO_3	-	7	reduced weight gain	Wahlstrom et al. (1956)
14 kg	Na_2SeO_3	-	10	reduced weight gain	Wahlstrom et al. (1956)
13 kg	Na_2SeO_3	-	11	reduced weight gain	Wahlstrom et al. (1956)
15 kg	Na_2SeO_3	-	10	reduced reproductive success	Wahlstrom and Olson (1959)
16-19 kg	Na_2SeO_3	-	24	Anorexia, death	Miller and Schoening (1938)

Table 5-2
Dietary toxicity studies on dogs from NRC (1980).

Age	Chemical Form	Concentration (mg/kg) NOAEL	LOAEL	Effect	Reference
60 days	Seleniferous corn	-	7.2	Decreased weight gain	Rhian and Moxon (1943)
150 days	Na_2SeO_3	-	10	Decreased weight gain	Rhian and Moxon (1943)
72 days	Seleniferous corn	-	20	Decreased weight gain	Rhian and Moxon (1943)
"young"	Na_2SeO_3	-	20	Decreased weight gain	Moxon (1937)
"young"	Seleniferous corn	-	20	Decreased weight gain	Moxon (1937)

For all of the studies on presented above, it is unclear whether the dietary concentrations are provided as ww or dw. Thus, the units are given only as mg/kg.

Mahan (2001) summarizes the existing studies on pigs by stating that “chronic selenosis generally occurs when diets or feedstuffs contain 5 to 20 ppm Se.”

5.3 Discussion

No selenium toxicity studies have been performed on marine mammals. Therefore, it was assumed that toxicity studies on other mammals would be representative of marine mammals. Among the mammals that have been studied, it was assumed that pigs and dogs are more likely to be representative of the marine mammals of concern in the North Bay than studies using ruminants and rodents. This is because ruminants and rodents have reducing conditions in their gastro-intestinal tracts that may reduce the bioavailability of selenium relative to the gastrointestinal tracts of pigs, dogs, seals, sea lions, and otters.

The studies reviewed by the NRC (1980, 2005) indicate that effects in pigs were not observed at dietary concentrations of less than 7 mg/kg (Table 5-1). In *Nutrient Requirements of Swine* (NRC 1998), a study that was not reviewed in the *Mineral Tolerance of Animals* (NRC 1980, 2005) was cited as causing adverse effects in pigs at 5 mg/kg. Mahan (2001) also states that effects have been observed in pigs at feed concentrations at 5 mg/kg. Thus, it appears that the lowest reported dietary concentration at which selenium has been shown to affect pigs is approximately 5 mg/kg.

The studies reviewed by the NRC (1980) on dogs did not administer dietary concentrations below 7.2 mg/kg. Since adverse effects were observed at that dietary concentration, it is not possible to determine the an actual effect threshold in dogs.

A point worthy of consideration is whether the type of food consumed affects the bioavailability and/or toxicity of selenium. The pig studies reviewed above would likely have used a feed that mainly consisted of corn and soybeans (NRC 1998), whereas seals, sea lions, and river otters are entirely carnivorous. Among pigs, "*animal Se sources generally have a lower (<25%) absorption rate than Se from plant tissue. However, Se absorption is influenced by the type of animal product being fed. For example, the absorption of Se from bovine milk is high and equivalent to that of selenite, whereas Se in meat and bone meal or poultry by-products is ≤20%*" (Mahan 2001). This implies that the bioavailability of selenium is likely to be lower in the diets of marine mammals than pigs and that the dietary screening level for selenium may need to be adjusted upwards of 5 mg/kg for marine mammals.

Allowable limits in feed

"In 1982, the FDA approved the addition of 0.3 ppm of selenium to diets for pigs up to 20 kg, because 0.1 ppm of added selenium does not always prevent deficiency signs in weanling pigs. The current regulation allows up to 0.3 ppm of selenium in the diet for all pigs" (NRC 1998).

Nutritional requirements

It should also be remembered that selenium is a micro-nutrient. When mammals do not have enough selenium in their diet, selenium deficiency results; i.e., edema, hepatic necrosis, poor growth, white muscle disease, mulberry heart disease, impaired reproduction, reduced milk production, impaired immune response, and mortality (NRC 1998). Due to the economic importance of the pig farming industry, the nutritional requirements to maintain an adequate growth rate and normal metabolism of pigs have been established by the NRC (1998; see Table 5-3 below) and Mahan (2001).

Table 5-3
Selenium nutritional requirements for commercially reared pigs (mg/kg-dw) by body weight (kg).

	NRC (1998)					Mahan (2001)			
Body weight (kg)	**3-10**	**10-20**	**20-120**	**125+**		**5-20**	**20-60**	**60+**	**-**
growing pigs	0.33	0.28	0.17			0.30	0.20	0.15	
gestating female				0.17					0.3
lactating female				0.17					0.3
Sexually active boars				0.17					

Maximum tolerable limits

The NRC (2005) defines the maximum tolerable level as the concentration "…of a mineral is the dietary level that, when fed for a defined period of time, will not impair animal health and performance." The NRC (2005) has set a maximum tolerable level of selenium in feed for pigs of 4 mg/kg (wet weight vs. dry weight not specified). The NRC (1998) also provides the following discussion of the maximum tolerable limit for selenium in pig feed:

"When fed to growing swine as sodium selenite, sodium selenate, selenomethionine, or seleniferous corn, selenium does not produce toxicity at levels of less than 5 ppm. In some cases, however, a level of 5 ppm and levels from 7.5 to 10 ppm have produced toxicity. Signs of toxicity include anorexia, hair loss, fatty infiltration of the liver, degenerative changes in the liver and kidney, edema, occasional separation of hoof and skin at the coronary band, and symmetrical, focal areas of vacuolation and neuronal necrosis."

Future research and data gaps

Several data gaps and areas where further research may be beneficial to the Water Board were identified during the evaluation of selenium toxicity to marine mammals in the North Bay, as follows:

1. Toxicity studies have not been performed on any of the marine mammals that reside in the North Bay. Thus, this report has assumed that toxicity studies on pigs and dogs are representative of harbor seals, sea lions, and river otters. The marine mammals in the North Bay may be more or less sensitive than pigs and dogs.
2. Toxicity studies on pigs have used a diet based on vegetable matter. The bioavailability of selenium in such diets is higher than in animal matter based diets. This means that the bioavailability of selenium in the diets of marine mammals may be lower than the in the toxicity studies that have been performed on pigs.
3. For this report, only the secondary literature was reviewed. A thorough examination of the primary literature (as was done for fish and birds) may prove more informative.
4. A detailed re-analysis of the published toxicity data for pigs using, for example, biphasic regressions (Beckon 2007, Beckon et al. 2008) would provide a greater level of confidence in estimates of the threshold for toxic effects of selenium vs. a LOAEL, NOAEL, or effect threshold. However, if only the treatment means are available for the analyses, it should be remembered that this type of analysis will mask the true variability in the data.
5. For harbor seals and sea lions, the collection and analysis of blood, hair, and blubber samples appears to be fairly routine. These types of samples can be collected without killing the seal/sea lion. It might prove useful to examine the dietary toxicity literature for pigs and develop relationships between dietary selenium levels and blood, fat, and/or hair levels in pigs that may be used to evaluate data collected from seals and sea lions in the North Bay.
6. The selenium toxicity studies performed to date have used selenite, selenomethionine, and selenized yeast. The degree to which the dietary formulations used in the toxicity studies reflects the speciation of selenium

speciation in the food of marine mammals of concern in the North Bay is not very well known. Since the speciation of selenium affects its toxicity, this may be an important consideration.

7. The secondary reviews do not detail whether the toxicity studies performed on pigs measured the selenium concentration in feed as ww or dw. This adds an additional layer of uncertainty.

5.4 CONCLUSIONS

Exposure to elevated levels of selenium can cause adverse effects in mammals. Based upon the data presented here, there are several options for selenium screening values protective of marine mammals. Those options are as follows (all in units of mg/kg):

1. The maximum tolerable limit recommended by that NRC (2005): 4
2. Lowest concentration at which adverse effects have been reported: 5

These proposed screening values are well in excess of the dietary requirements for pigs (which are assumed here to be representative of marine mammals) of approximately 0.3 mg/kg-dw (Table 5-3).

6 Issues Affecting The Choice of Screening Values

This report has presented several options for screening values protective of different receptor groups. Here, we discuss issues related to selecting a screening value for use in the TMDL process.

Derivation of screening values

In Section 1, multiple methods were identified that have been used in the toxicity literature to derive screening values, including:

- LOAELs
- Effect thresholds
- Species mean chronic values
- EC01 or EC10
- Species sensitivity distributions

Further, there are multiple means of calculating an effect concentrations (i.e., an EC01 or EC10) or a species sensitivity distribution. This means that different mathematical techniques can be applied to the same data to derive different screening values.

Determining which method is the best is no easy matter, as each method has its own limitations. Most of the selenium toxicity data examined in this review was collected from experiments designed to be analyzed using an ANOVA; i.e., there are several groups of animals in an experiment and each group is exposed to a different selenium dose. These types of experiments produce a NOAEL and a LOAEL; i.e., the highest selenium dose at which no adverse effects were observed and the lowest selenium dose at which adverse effects were observed.

Some authors have re-analyzed the treatment means from these kind of experiments using a regression analysis (e.g., logistic, hockey stick, or biphasic) to obtain a better understanding of the threshold for toxic effects than is provided by the NOAEL and LOAEL (e.g., Adams 2003, Ohlendorf 2003, 2007, Beckon 2007, 2008). However, in analyzing only the treatment means, significant variation is lost and the toxicity thresholds derived are uncertain.

Toxicity experiments designed to be analyzed using regression analyses have the potential to be much more powerful than toxicity tests designed to be analyzed using an ANOVA; i.e., "the purposes of accurately determining risk and maximizing benefit are more efficiently served by the allocation of limited experimental resources to a greater range of dose levels rather than to multiple replicates at a small number of dose levels. The latter, a legacy of traditional hypothesis testing, is relatively poorly suited for fitting and comparing mathematical models of the dose-response relationship" (Beckon et al. 2008). Therefore, it is recommended that future toxicity studies be designed with a regression analysis in mind.

When using an effect concentration (EC) or a species sensitivity distribution, one must also decide on what level to protect in order to derive the screening value. For effect thresholds, it is common to set the threshold at a 1, 10, or even 20% effect. For species sensitivity distributions, it is common to choose a value that is protective of 95% of the species evaluated. Choosing what threshold to use should be related to what level may actually impact populations in the field, yet information on that is scant. Further, when comparing screening values derived using either of these methods, one must also consider the confidence limits; e.g., is an EC10 of 1.8 significantly different from an EC20 of 2.5?

Uncertainties in toxicity testing

There are some additional sources of uncertainty associated with toxicological studies reviewed here that should be considered when interpreting the results to set regulatory standards, including the following:

- Responses to selenium differ among species and life-stage within the same species. However, results for one species of life-history stage are used to assess the effects on other species and life stages. For example, the only toxicity data available for white sturgeon were derived from short-term chronic (8-week) exposures to juvenile fish, but this fish is long-lived and does not reach sexual maturity until they are 10 years of age or more.

- In short-term toxicity tests performed on fish in the laboratory, the concentrations in the tissue may not have reached steady state. Thus, screening values derived from short term studies where tissue concentrations have not yet reached equilibrium may underestimate the tissue level that causes toxicity. However, current recommendations are to use fish tissue concentrations as screening values (Hamilton 2002, 2003, 2004, USEPA 2004).

- There is a lack of data linking the results of laboratory toxicity data to selenium concentrations and effects observed in the field. Even though effects have been demonstrated in the laboratory, it is unknown whether these effects will translate to population level effects in the field; i.e., population declines.

Current selenium toxicity testing efforts

At a recent meeting of the North American Industry Selenium Working Group (November 16, 2007) several new selenium toxicity studies were announced. These studies include the following:

- J.R. Simplot will spend approximately $500K for selenium toxicity testing with brown trout, Yellowstone cutthroat trout, and possibly a third species.

- USEPA has performed a repeat of the bluegill toxicity test done by Lemly (1993a) upon which the USEPA (2004) based its draft ambient water quality criterion. Toxicity was found at higher concentrations that observed by Lemly (1993a). These results have not yet been published.

- The North American Industry Selenium Working Group may be funding relatively small studies on "site-specific assessments of selenium bioaccumulation in aquatic systems" and "tissue endpoint assessment." However, the details of these studies are unknown.

- Cameco is performing "water bird research in Saskatchewan including assessing food, productivity, and egg [Se] for tree swallows."

The research that is currently in progress described above is all on freshwater ecosystems. So, although the research is of general interest, it will not address the issues of greatest concern to the Board; i.e., the toxicity of selenium of brackish/marine ecosystems and the toxicity of selenium to the species of concern in the North Bay. Further, the current research does not appear to have been designed for use with regression analyses, meaning the NOAELs and LOAELs will be provided and deriving an EC20 will still be associated with some uncertainty.

Conclusions

Ultimately, given importance of selenium to the water resources of California, and the history of its effects, it is surprising that so little toxicity data on the species of concern in California is available. The methods used in selenium toxicity testing to date do not provide regulatory entities with a high degree of confidence in deriving toxicity thresholds and/or screening values. Lastly, field validation of selenium toxicity studies should also be performed to increase confidence in any screening levels derived from the existing toxicity literature.

7 ISSUES AFFECTING IMPLEMENTATION

When sampling invertebrates, fish, bird eggs, or other biological media to compare to the selenium screening values proposed in the earlier sections of this report, there are several issues that should be considered to maximize the use of the data. This section of the report provides a brief discussion of some of the more important issues. Some of these issues have been considered by various authors (e.g., Lemly and Skorupa 2007, McDonald and Chapman 2007, USEPA 2000, 2006).

Spatial variability

The concentration of selenium in the waters of the North Bay is expected to differ depending upon where samples are collected. Near selenium inputs (e.g., refineries, some creeks, and the San Joaquin River), selenium concentration in the water column are expected to be higher. This should result in higher concentrations for the biota that inhabit that area, providing that they have a relatively small range. For example, a clam may move only a few feet within its lifetime, but a sea lion may travel thousands of miles. Therefore, if there is an area where there is a localized input of selenium, elevated selenium levels would be expected in biota with a small range (e.g., the clam) but not necessarily in biota with a large range (e.g., the sea lion). This suggests that samples should be collected from areas near known selenium inputs from biota that are most likely to be impacted. To determine the extent of impacts, a number of locations should be sampled, not just areas near selenium inputs.

Background

Since selenium is a naturally occurring element, it is expected to be present at some concentration in all waters, soils, and sediments in the North Bay. Therefore, in addition to examining the spatial variability of selenium in the North Bay, it may be desirable to establish an area (or areas) that are representative of unimpacted (or reference) conditions. Then, biota may be sampled from that area and selenium concentrations in the tissues of biota from the background location can be compared to the samples collected from the North Bay to determine what areas are impacted. If this technique is used, it is advised that statistically robust techniques be used to compare sample locations, such as the t-test, ANOVA, or Kruskall-Wallis test.

Temporal variability

Concentrations of selenium in tissues may vary with the time of year. USEPA (2006) recommends that fish samples be collected during the same time of year during each sample event to avoid potential temporal effect.

Comparing measured concentrations to screening values

USEPA (2006) "… recommends using the [one sample] t-test to determine whether the mean concentration of mercury in composite fish tissue samples exceeds the screening value. This involves a statistical comparison of the mean of all fish tissue data to the criterion. EPA recommends that this procedure also be used for determining impairment." This technique is also applicable to selenium (without the compositing). To use a one-sample t-test, the screening value is compared against the mean concentration for all of the biota samples of the same species collected from the location/time being evaluated. The results of the test can be used to determine whether the measured concentrations significantly exceed the screening value or not.

Another approach that can also be used is to compare the 95% upper confidence limit on the mean (UCL_{95}) to the screening value. If the calculated UCL_{95} does not exceed the screening value, then no action is necessary. USEPA (2007b) provides free statistical software that can be used to calculate a UCL_{95}s for selenium data in biological samples.

Lastly, it is also possible to simply compare measured concentrations to the screening values. This method is likely to result in more exceedances than using a one sample t-test or a UCL_{95}. In that respect, comparing individual concentrations to the screening value may be more "sensitive" but it is not statistically valid.

Food webs

It has been demonstrated that among the fish species in the North Bay that those that feed on clams, or are part of a food web that starts with clams, have higher selenium tissue concentrations (Stewart et al. 2004). This is because the concentrations of selenium in one of the more abundant clams in the North Bay, *Potamocorbicula amurensis*, are much higher than many of the other invertebrates that fish feed on (Stewart et al. 2004). Similarly, it would be expected that birds that feed on this species of clam have higher selenium exposures than other bird species. In addition, it is generally expected that fish and birds that feed on biota in the sediments of the Bay would have greater selenium exposures than fish and birds that feed in the open water column; i.e., the Delta smelt only feed on zooplankton near the top of the water column and would, therefore, be expected to be exposed to less selenium than fish that feed in the sediments, such as sturgeon. However, it is possible that species that have a lower potential for exposure may be more sensitive to selenium. Thus, the other species in the North Bay should not be ignored.

Size of specimens

Data collected by the USGS indicate that as the size of Sacramento splittail increases over their lifetime, the fish's dietary habits change and that this affects their bioaccumulation of selenium. In addition, the concentrations of many metals increase with the size of the specimen sampled. For selenium, this does not always appear to be the case, but the potential effect of body size should be accounted for when collecting biota samples. Thus, body size (i.e., length and weight) should always be measured and data should be corrected for any relationship with size/weight (Tremblay et al. 1998, Wente 2004).

Frequency of collection

USEPA (2006) recommends that fish samples be collected at least once every two years to determine compliance with the fish tissue criterion for methylmercury. If a monitoring strategy for the North Bay is implemented, it may be appropriate to collect samples annually, at least initially.

Compositing

When collecting fish samples to support fish consumption advisories, USEPA (2000, 2006) recommends the collection of composite samples. What this means in that samples are collected from several fish individuals, but then the samples are homogenized and the laboratory analyzes only the single composite sample. This approach is designed to minimize costs and to provide an indication of the average concentration. However, compositing biota samples makes evaluating relationships between 1) age, sex, size, time of year and 2) selenium concentration more difficult. Further, the costs for low level selenium analyses in tissues have fallen in recent years. Therefore, it is recommended that biota samples should not be composited, at least for organisms where it is possible to obtain samples large enough for analysis from a single individual (i.e., 2 g).

Sample size
If the Water Board elects to use the statistical techniques recommended above, then it is recommended that a minimum of eight samples be collected for a species during each sampling event. This will provide the necessary sample size to perform both background comparisons and the calculation of a UCL_{95}. This is consistent with USEPA (2007b) recommendations.

Fish tissue screening value protective of human health
The State of California (OEHHA 2006) has calculated a screening value for selenium in fish tissue that is protective of fishermen (i.e., the human consumption fish). The screening value was calculated using toxicity data from USEPA (2007c). It should be noted that the calculations that the State of California (OEHHA 2006) used assume a 100% bioavailability of selenium in fish tissues to humans. However, as stated in the section on the toxicity of selenium to marine mammals, the bioavailability of selenium in animal tissues is generally lower (<25%) (Mahan 2001). This may need to be evaluated in more detail prior to implementing the fish tissue screening value protective of human health in the North Bay.

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APPENDIX A

WET WEIGHT TO DRY WEIGHT CONVERSIONS

Concentrations of selenium in tissues and animal feed are reported in either wet weight (ww) or dry weight (dw). Unfortunately, the two measurements are not equivalent. The selenium concentrations that were used in the analyses presented earlier in this report are presented in their original units in Table A-1. All of the selenium concentrations reported in Table A-1 were converted to dry weight using either the percent moisture reported in the original publication or the default percent moisture of 75% (USEPA 1993) for studies on fish where the percent moisture was not reported. The percent moisture used in the calculations is presented in Table A-1 and the dry weight selenium concentrations are reported in Table A-2. The formula used to covert wet weight to dry weight is as follows:

$$\text{Dry weight} = \text{wet weight} \times \frac{100}{100 - \text{percent moisture}}$$

Additionally, not all studies measured selenium in all tissue types. USEPA (2004) provides equations to convert selenium concentrations in fish muscle and liver to whole body concentrations. The data that USEPA (2004) presented was also used to derive linear regression equations to convert selenium concentrations measured in one tissue type to another tissue type for all fish, except white sturgeon. The equations used are provided below and are given in order of preference used to estimate tissue concentrations.

1. Selenium in fish muscles to whole body (USEPA 2004):

$$Se_{wholebody} = \exp\left[0.1331 + \left(0.8937 \times \ln\left(Se_{muscle}\right)\right)\right]$$

2. Selenium in fish liver to whole body (USEPA 2004):

$$Se_{wholebody} = -0.2609 + \left(0.3071 \times Se_{liver}\right)$$

3. Selenium in whole fish bodies to fish livers ($p < 0.0001$, $r^2 = 0.73$):

$$Se_{liver} = \exp\left[1.6475 + \left(0.7628 \times \ln\left(Se_{wholebody}\right)\right)\right]$$

4. Selenium in whole fish bodies to fish muscle ($p < 0.0001$, $r^2 = 0.96$):

$$Se_{muscle} = \exp\left[0.0324 + \left(1.031 \times \ln\left(Se_{wholebody}\right)\right)\right]$$

For white sturgeon, TetraTech obtained the raw data from the study conducted by Tashjian et al. (2006) and used that data to calculate the following regression equation ($F_{1,22} = 222.42$, Adjusted $R^2 = 0.91$, $p < 0.00001$):

$$Se_{wholebody} = \exp\left[0.9586 + \left(0.6206 \times \ln\left(Se_{muscle}\right)\right)\right]$$

This regression equation was used to convert the muscle EC10 derived by Beckon to a whole body concentration as well as the muscle NOAEL and LOAEL from Linville (2006) to whole body concentrations.

For the selenium toxicity studies on birds, only the concentration in the feed given to the birds was converted from wet weight to dry weight. That is presented in Table A-4 for toxicity studies and A-5 for production studies.

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Table A-1
Fish Tissue Concentration Data
Measured Selenium Concentrations (mg/kg or ug/g)

		Water		Exposure	Kidney Se (mg/kg)		Liver Se (mg/kg)		Muscle Se (mg/kg)		Whole Body Se (mg/kg)					Effect		Used in
Fish	**Study**	**Type**	**Life stage**	**Route**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**dw/ww**	**%moisture[1]**	**Notes**	**Major**	**Minor**	**SMCV**
Bluegill	Cleveland (1993)	fresh	Juvenile	Water	-	-	-	-	-	-	3.0	3.4	dw	-	2	X		
Bluegill	Cleveland (1993)	fresh	Juvenile	Diet	-	-	-	-	-	-	4.7	7.6	dw	-	2	X		X
Bluegill	Coyle et al. (1993)	fresh	Adult	Water/Diet	-	-	-	-	-	-	10	19	dw	-	2,4	X		X
Bluegill	Coyle et al. (1993)	fresh	egg	Water/Diet	-	-	-	-	-	-	23	41	dw	-	2,4	X		
Bluegill	Doroshov et al. (1992)	fresh	Adult	Diet	-	-	12.3	26.0	5.8	10.4	-	-	dw	-	-	X		X
Bluegill	Finley (1985)	fresh	adult	Diet	-	-	5.0	8.5	2.06	5.10	-	-	ww	75	-	X		
Bluegill	Gillespie and Baumann (1986)	fresh	Fry/larvae	Natural	-	-	-	-	-	-	3.1	28.2	dw	-	-	X		
Bluegill	Gillespie and Baumann (1986)	fresh	Adult	Natural	-	-	-	-	-	-	0.4	5.9	ww	75	-	X		
Bluegill	Hermanutz et al. (1992)	fresh	adult	Water/Diet	-	-	1.5	9.3	0.3	1.8	-	-	ww	75	-	X		X
Bluegill	Lemly (1993a); Skorupa et al. 2004	fresh	Juvenile	Water/Diet	-	-	-	-	-	-	1.3	5.8	dw	-	-	X		X
Bluegill	Sorensen et al. (1984)	fresh	Adult	Natural	-	-	1.3	7.0	1.3	2.3	-	-	ww	75	-	X		
channel catfish	Doroshov et al. (1992)	fresh	Adult	Injection	-	-	7.3	12.5	-	-	-	-	dw	-	-			
channel catfish	Gatlin and Wilson (1984)	fresh	fingerling	Diet	-	-	-	-	3.0	3.5	-	-	dw	-	-	X		X
chinook salmon	Hamilton et al. (1990)	fresh	larvae	Diet	-	-	-	-	-	-	5.4	10.8	dw	-	6	X		X
chinook salmon	Hamilton et al. (1990)	brackish	juvenile	Diet	-	-	-	-	-	-	12.6	23.2	dw	-	6	X		X
fathead minnow	Dobbs et al. (1996)	fresh	larvae	Water/Diet	-	-	-	-	-	-	-	62	dw	-	-	X		X
fathead minnow	Ogle and Knight (1989)	fresh	juvenile	Diet	-	-	-	-	5.7	6.6	5.4	6.6	dw	-	-	X		X
fathead minnow	Schultz and Hermanutz (1990)	fresh	egg	Water/Diet	-	-	-	-	-	-	0.3	3.9	ww	80	-	X		X
green sunfish	Lemly (1993b)	fresh	Adult	Natural	-	-	-	-	-	-	12.4	14.7	dw	-	-	X		
rainbow trout	Hicks et al (1984)	fresh	juvenile	Diet	3.0	10.7	38.3	49.3	-	-	-	-	ww	75	-	X		X
rainbow trout	Hilton and Hodson (1983)	fresh	juvenile	Diet	0.7	3.8	0.6	21.0	-	-	0.2	1.0	dw	-	3, 4		X	
rainbow trout	Hilton and Hodson (1983)	fresh	juvenile	Diet	3.8	7.7	21.0	71.7	-	-	1.0	4.0	dw	-	3, 4	X		X
rainbow trout	Hilton et al. (1980)	fresh	juvenile	Water/Diet	9	37	42.0	95.0	-	-	-	-	dw	-	2	X		X
rainbow trout	Holm et al. (2005)	fresh	adult	Natural	-	-	-	-	-	1.8	-	-	ww	75	-	X		
rainbow trout	Holm et al. (2005)	fresh	egg	Natural	-	-	-	-	-	-	-	3.5	ww	61	-	X		
rainbow trout	Hunn et al (1987)	fresh	Fry/larvae	Water	-	-	-	-	-	-	0.5	0.9	dw	-	4		X	
rainbow trout	Hunn et al (1987)	fresh	Fry/larvae	Water	-	-	-	-	-	-	0.6	1	dw	-	4	X		
rainbow trout	Vidal et al (2005)	fresh	Fry/larvae	Diet	-	-	-	-	-	-	0.3	0.6	ww	75	-	X		X
Redear sunfish	Sorensen et al. (1988)	fresh	Adult	Natural	2.1	7.6	-	-	0.3	5.0	-	-	ww	75	5	X		
Sacramento splittail	Teh et al. (2004)	fresh	juvenile	Diet	-	-	23.0	26.8	10.1	15.1	-	-	dw	-	-	X		X
Striped Bass	Coughlan and Velte (1989)	fresh	Adult	Diet	-	-	-	-	1.1	3.5	-	-	ww	75	-	X		
white sturgeon	Linville (2006)	fresh	Adult	Diet	-	-	1.4	10.4	1.3	12.1	-	-	dw	-	-	X		X
white sturgeon	Tashjian et al. (2006)	fresh	juvenile	Diet	30.9	51.7	22.0	37.4	22.9	36.8	14.7	22.5	dw	-	-	X		X

Notes:

1 - a value of 75% is the default assumpttion (USEPA 19930

2 - concentration estimated from figures

3 - data from the low carbohydrate diet were not used as the authors reported that reduced weight of fish on this diet was likely due to food

4 - dry weight assumed. Not explicilty stated whether dw or ww

5 - muscle concentration calculated using regression equation from Sorensen et al. (1982); i.e., muscle concentation = kidney concentration x 0.835 -1.375

6 - excludes treatment where fish were exposed to water from the San Luis Drain

Table A-2
Wet Weight to Dry Weight Conversion for Fish Tissue Concentration Data
Selenium Concentrations in Dry Weight (mg/kg-dw or ug/g-dw)

		Water		Exposure	Kidney Se (mg/kg)		Liver Se (mg/kg)		Muscle Se (mg/kg)		Whole Body Se (mg/kg)		Effect		Used in
Fish	**Study**	**Type**	**Life stage**	**Route**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**Major**	**Minor**	**SMCV**
Bluegill	Cleveland (1993)	fresh	Juvenile	Water	-	-	-	-	-	-	3.0	3.4	X		
Bluegill	Cleveland (1993)	fresh	Juvenile	Diet	-	-	-	-	-	-	4.7	7.6	X		X
Bluegill	Coyle et al. (1993)	fresh	Adult	Water/Diet	-	-	-	-	-	-	10.0	19.0	X		X
Bluegill	Coyle et al. (1993)	fresh	egg	Water/Diet	-	-	-	-	-	-	23.0	41.0	X		
Bluegill	Doroshov et al. (1992)	fresh	Adult	Diet	-	-	12.3	26.0	5.8	10.4	-	-	X		X
Bluegill	Finley (1985)	fresh	adult	Diet	-	-	20.0	33.9	8.2	20.4	-	-	X		
Bluegill	Gillespie and Baumann (1986)	fresh	Fry/larvae	Natural	-	-	-	-	-	-	3.1	28.2	X		
Bluegill	Gillespie and Baumann (1986)	fresh	Adult	Natural	-	-	-	-	-	-	1.5	23.6	X		
Bluegill	Hermanutz et al. (1992)	fresh	adult	Water/Diet	-	-	6.0	37.2	1.2	7.2	-	-	X		X
Bluegill	Lemly (1993a); Skorupa et al. 2004	fresh	Juvenile	Water/Diet	-	-	-	-	-	-	1.3	5.8	X		X
Bluegill	Sorensen et al. (1984)	fresh	Adult	Natural	-	-	5.2	28.0	5.2	9.2	-	-	X		
channel catfish	Doroshov et al. (1992)	fresh	Adult	Injection	-	-	7.3	12.5	-	-	-	-	X		X
channel catfish	Gatlin and Wilson (1984)	fresh	fingerling	Diet	-	-	-	-	3.0	3.5	-	-	X		X
chinook salmon	Hamilton et al. (1990)	fresh	larvae	Diet	-	-	-	-	-	-	5.4	10.8	X		X
chinook salmon	Hamilton et al. (1990)	brackish	juvenile	Diet	-	-	-	-	-	-	12.6	23.2	X		X
fathead minnow	Dobbs et al. (1996)	fresh	larvae	Water/Diet	-	-	-	-	-	-	-	62.0	X		X
fathead minnow	Ogle and Knight (1989)	fresh	juvenile	Diet	-	-	-	-	5.7	6.6	5.4	6.6	X		X
fathead minnow	Schultz and Hermanutz (1990)	fresh	egg	Water/Diet	-	-	-	-	-	-	1.6	19.6	X		X
green sunfish	Lemly (1993b)	fresh	Adult	Natural	-	-	-	-	-	-	12.4	14.7	X		
rainbow trout	Hicks et al (1984)	fresh	juvenile	Diet	12.0	42.8	153.2	197.2	-	-	-	-	X		X
rainbow trout	Hilton and Hodson (1983)	fresh	juvenile	Diet	0.7	3.8	0.6	21.0	-	-	0.2	1.0		X	
rainbow trout	Hilton and Hodson (1983)	fresh	juvenile	Diet	3.8	7.7	21.0	71.7	-	-	1.0	4.0	X		X
rainbow trout	Hilton et al. (1980)	fresh	juvenile	Water/Diet	9.0	37.0	42.0	95.0	-	-	-	-	X		X
rainbow trout	Holm et al. (2005)	fresh	adult	Natural	-	-	-	-	-	7.2	-	-	X		
rainbow trout	Holm et al. (2005)	fresh	egg	Natural	-	-	-	-	-	-	-	9.0	X		
rainbow trout	Hunn et al (1987)	fresh	Fry/larvae	Water	-	-	-	-	-	-	0.5	0.9		X	
rainbow trout	Hunn et al (1987)	fresh	Fry/larvae	Water	-	-	-	-	-	-	0.6	1.1	X		
rainbow trout	Vidal et al (2005)	fresh	Fry/larvae	Diet	-	-	-	-	-	-	1.2	2.3	X		X
Redear sunfish	Sorensen et al. (1988)	fresh	Adult	Natural	8.2	30.5	-	-	1.3	20.0	-	-	X		
Sacramento splittail	Teh et al. (2004)	fresh	juvenile	Diet	-	-	23.0	26.8	10.1	15.1	-	-	X		X
Striped Bass	Coughlan and Velte (1989)	fresh	Adult	Diet	-	-	-	-	4.4	14.0	-	-	X		
white sturgeon	Linville (2006)	fresh	Adult	Diet	-	-	1.4	10.4	1.3	12.1	-	-	X		X
white sturgeon	Tashjian et al. (2006)	fresh	juvenile	Diet	30.9	51.7	22.0	37.4	22.9	36.8	14.7	22.5	X		X

Table A-3
Conversion of Fish Tissue Concentration Data Among Tissue Types
Selenium Concentrations in Dry Weight (mg/kg-dw or ug/g-dw)

		Water		**Exposure**	**Kidney Se (mg/kg)**		**Liver Se (mg/kg)**		**Muscle Se (mg/kg)**		**Whole Body Se (mg/kg)**		**Effect**		**Used in**
Fish	**Study**	**Type**	**Life stage**	**Route**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**NOAEL**	**LOAEL**	**Major**	**Minor**	**SMCV**
Bluegill	Cleveland (1993)	fresh	Juvenile	Water	-	-	12.0	13.2	3.2	3.6	3.0	3.4	X		
Bluegill	Cleveland (1993)	fresh	Juvenile	Diet	-	-	16.8	24.5	5.1	8.4	4.7	7.6	X		X
Bluegill	Coyle et al. (1993)	fresh	Adult	Water/Diet	-	-	30.1	49.1	11.1	21.5	10.0	19.0	X		X
Bluegill	Coyle et al. (1993)	fresh	egg	Water/Diet	-	-	-	-	-	-	23.0	41.0		X	
Bluegill	Doroshov et al. (1992)	fresh	Adult	Diet	-	-	12.3	26.0	5.8	10.4	5.5	9.3	X		X
Bluegill	Finley (1985)	fresh	adult	Diet	-	-	20.0	33.9	8.2	20.4	7.5	16.9	X		
Bluegill	Gillespie and Baumann (1986)	fresh	Fry/larvae	Natural	-	-	12.3	66.3	3.3	32.3	3.1	28.2	X		
Bluegill	Gillespie and Baumann (1986)	fresh	Adult	Natural	-	-	7.0	58.0	1.5	26.9	1.5	23.6		X	
Bluegill	Hermanutz et al. (1992)	fresh	adult	Water/Diet	-	-	6.0	37.2	1.2	7.2	1.3	6.7	X		X
Bluegill	Lemly (1993a); Skorupa et al. 2004	fresh	Juvenile	Water/Diet	-	-	6.3	19.9	1.4	6.3	1.3	5.8	X		X
Bluegill	Sorensen et al. (1984)	fresh	Adult	Natural	-	-	5.2	28.0	5.2	9.2	5.0	8.3	X		
channel catfish	Doroshov et al. (1992)	fresh	Adult	Injection	-	-	7.3	12.5	2.1	3.8	2.0	3.6	X		X
channel catfish	Gatlin and Wilson (1984)	fresh	fingerling	Diet	-	-	-	-	3.0	3.5	3.1	3.5	X		X
chinook salmon	Hamilton et al. (1990)	fresh	larvae	Diet	-	-	18.8	31.9	5.9	12.0	5.4	10.8	X		X
chinook salmon	Hamilton et al. (1990)	brackish	juvenile	Diet	-	-	35.9	57.2	14.1	26.4	12.6	23.2	X		X
fathead minnow	Dobbs et al. (1996)	fresh	larvae	Water/Diet	-	-	-	121.0	-	72.8	-	62.0	X		X
fathead minnow	Ogle and Knight (1989)	fresh	juvenile	Diet	-	-	18.8	21.9	5.7	6.6	5.4	6.6	X		X
fathead minnow	Schultz and Hermanutz (1990)	fresh	egg	Water/Diet	-	-	-	-	-	-	1.6	19.6	X		X
green sunfish	Lemly (1993b)	fresh	Adult	Natural	-	-	35.4	40.3	13.8	16.5	12.4	14.7	X		
rainbow trout	Hicks et al (1984)	fresh	juvenile	Diet	12.0	42.8	153.2	197.2	-	-	46.8	60.3	X		X
rainbow trout	Hilton and Hodson (1983)	fresh	juvenile	Diet	0.7	3.8	0.6	21.0	0.2	1.0	0.2	1.0	X		
rainbow trout	Hilton and Hodson (1983)	fresh	juvenile	Diet	3.8	7.7	21.0	71.7	1.0	4.3	1.0	4.0	X		X
rainbow trout	Hilton et al. (1980)	fresh	juvenile	Water/Diet	9.0	37.0	42.0	95.0	-	-	12.6	28.9	X		X
rainbow trout	Holm et al. (2005)	fresh	adult	Natural	-	-	-	-	-	7.2	-	6.7	X		
rainbow trout	Holm et al. (2005)	fresh	egg	Natural	-	-	-	-	-	-	-	9.0	X		
rainbow trout	Hunn et al (1987)	fresh	Fry/larvae	Water	-	-	3.2	4.7	0.5	0.9	0.5	0.9	X		
rainbow trout	Hunn et al (1987)	fresh	Fry/larvae	Water	-	-	3.7	5.5	0.7	1.1	0.6	1.1	X		
rainbow trout	Vidal et al (2005)	fresh	Fry/larvae	Diet	-	-	6.1	9.9	1.3	2.5	1.2	2.3	X		X
Redear sunfish	Sorensen et al. (1988)	fresh	Adult	Natural	8.2	30.5	-	-	1.3	20.0	1.5	16.6	X		
Sacramento splittail	Teh et al. (2004)	fresh	juvenile	Diet	-	-	23.0	26.8	10.1	15.1	9.0	12.9	X		X
Striped Bass	Coughlan and Velte (1989)	fresh	Adult	Diet	-	-	-	-	4.4	14.0	4.3	12.1	X		
white sturgeon	Linville (2006)	fresh	Adult	Diet	-	-	1.4	10.4	1.3	12.1	3.1	12.3	X		X
white sturgeon	Tashjian et al. (2006)	fresh	juvenile	Diet	30.9	51.7	22.0	37.4	22.9	36.8	14.7	22.5	X		X

Note: shaded cells represent predicted concentrations. See text for explanation.

Table A-4
Wet Weight to Dry Weight Conversion for Bird Dietary Concentration Data
Measured Selenium Concentrations (mg/kg or ug/g)

				Measured Diet Se (mg/kg)				Diet Se (mg/kg-dw)		Effect	
Bird	**Study**	**Life stage**	**Chemical form**	**NOAEL**	**LOAEL**	**dw/ww**	**%moisture**	**NOAEL**	**LOAEL**	**Major**	**Minor**
Chicken	Elzubier and Davis (1988)	Chick	selenite	0.15	10	?	?	0.15	10	X	
Chicken (Hubbard)	Jensen (1976)	Chick	selenite	0.9	4.3	dw	-	0.9	4.3	X	
Chicken (leghorn)	Jensen (1976)	Chick	selenite	4.3	13.5	dw	-	4.3	13.5	X	
Chicken	Lowry and Baker (1989)	Chick	selenite; SeMet	0	15	?	?	0	15	X	
Chicken	Ort and Latshaw (1978)	Adult	selenite	3	5	?	?	3	5	X	
Common eider	Franson et al. (2007)	Adult	SeMet	20.6	57.7	dw	-	20.6	57.7	X	
Mallard	Albers et al. (1996)	Adult	SeMet	20	40	ww	10.5	22.3	44.7	X	
Mallard	Green and Albers 1996	Adult	SeMet	10	20	ww	10.5	11.2	22.3	X	
Mallard	Heinz and Hoffman, 1998	Adult	SeMet	0.4	8.8	ww	10	0.4	9.8	X	
Mallard	Heinz et al., 1987	Adult	selenite	10	25	ww	8.5	10.9	27.3	X	
Mallard	Heinz et al., 1987	Adult	SeMet	0	10	ww	8.5	0.0	10.9	X	
Mallard	Heinz et al., 1988	Duckling	selenite	10.1	20.1	ww	7	10.9	21.6	X	
Mallard	Heinz et al., 1988	Duckling	SeMet	10.1	20.1	ww	7	10.9	21.6	X	
Mallard	Heinz et al., 1989	Adult	SeMet	4.1	8.1	ww	10	4.6	9.0	X	
Mallard	Heinz et al., 1989	Adult	SeCysteine	10.1	-	ww	10	11.2	-	X	
Mallard	Heinz et al. (1996)	Duckling	SeMet	15.34	30.34	ww[1]	10	17.0	33.7	X	
Mallard	Hoffman and Heinz (1998)	Adult	SeMet	0.4	10.4	ww	10	0.4	11.6		X
Mallard	Hoffman et al. (1991)	Duckling	SeMet; low protein	0.2	15.2	ww	10	0.2	16.9	X	
Mallard	Hoffman et al. (1991)	Duckling	SeMet	15.2	60.2	ww	10	16.9	66.9	X	
Mallard	Hoffman et al. (1992a)	Duckling	SeMet	0.2	15.2	ww	10	0.2	16.9	X	
Mallard	Hoffman et al., 1992b	Duckling	SeMet	0.2	15.2	ww	8	0.2	16.5		X
Mallard	Hoffman et al., 1992b	Duckling	SeMet	15.2	60.2	ww	8	16.5	65.4	X	
Mallard	Hoffman et al., 1992b	Duckling	SeMet; low protein	15.2	60.2	ww	8	16.5	65.4	X	
Mallard	Hoffman et al., 1992b	Duckling	SeMet; low protein	0.2	15.2	ww	8	0.2	16.5		X
Mallard	Hoffman et al., 1992b	Duckling	SeMet; high protein	0.2	15.2	ww	8	0.2	16.5	X	
Mallard	Hoffman et al. (1996)	Duckling	SeMet, Se yeast, Se in wheat	0.2	15.2	ww[1]	10	0.2	16.9		X
Mallard	O'Toole and Raisbeck (1997)	Flightling	SeMet	10.32	25.32	ww	25	13.8	33.8	X	
Mallard	Stanley et al. (1994)	Adult	SeMet	0.37	6.5	dw	-	0.37	6.5	X	
Mallard	Stanley et al. (1996)	Adult	SeMet	3.5	7	ww	10	3.9	7.8	X	
Pheasant	Latshaw et al. (2004)	Adult	unknown	-	9.3	dw	-	-	9.3	X	
Screech owl	Wiemeyer and Hoffman (1996)	Adult	SeMet	8.8	30	dw	-	8.8	30	X	
Screech owl	Wiemeyer and Hoffman (1996)	Adult	SeMet	0.3	8.8	dw	-	0.3	8.8		X

Notes: 1 - a %moisture of 10 was assumed based on other studies by the same authors where a %moisture was specified.

Table A-5
Summary of Chicken Enhanced Production Studies
Measured Selenium Concentrations (mg/kg or ug/g)

Bird	Study	Life stage	Dietary form	Measured NOAEL (mg/kg)	dw/ww	%moisture	NOAEL (mg/kg-dw)	Effects: Survival	Effects: Weight	Effects: Egg output	Effects: Reproductive success
Chicken	Payne et al. (2005)	Adult	selenite	2.6	?	-	2.6			X	
Chicken	Payne et al. (2005)	Adult	Se-yeast	2.9	?	-	2.9			X	
Chicken	Ryu et al. (2005)	1 day	selenite	8.2	dw	-	8.2		X		
Japanese quail	Biswas et al. (2006)	1 day	selenite	1.2	?	-	1.2	X	X		
Japanese quail	Sahin and Kucuk (2001)	10 day	selenite	0.2	ww	10.32	0.2		X		

APPENDIX B
THRESHOLD VALUES FOR SELENIUM IN GREAT SALT LAKE

Threshold Values for Selenium in Great Salt Lake: Selections by the Science Panel

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The purpose of this technical memorandum is to provide a summary and documentation of the Science Panel's discussions relative to toxicity thresholds for exposure of birds to selenium at the Great Salt Lake. It is generally recognized that the most significant exposure of birds occurs through their diet, and that the best-documented and most readily-monitored effects are those on reproductive success (particularly egg hatchability). Thus, much of the focus of this technical memorandum is on those exposures and endpoints, because they can be most readily applied toward establishment of a site-specific water quality standard for selenium in the open waters of the Great Salt Lake.

Before the Science Panel meeting on November 29-30, 2006, I prepared a technical memorandum (Subject: Threshold Values for Selenium in Great Salt Lake; dated November 28) to provide the following:

- a summary of potential threshold values identified by Science Panel members for consideration in establishing a water quality standard for selenium in the open waters of the Great Salt Lake, and
- supporting documentation and literature provided by Panel members to be used as the basis of discussion by the Panel.

Bill Adams, Anne Fairbrother, Theresa Presser, and Joe Skorupa provided input concerning threshold values to be considered and sent supporting literature (either as citations or copies of publications), in addition to providing their views on the threshold values themselves. The entire Panel discussed that material and related information from other sources on November 30. From the available information, the Panel narrowed the ranges of values for bird diets and eggs to those listed in Tables 1 and 2 (Attachment A [tables modified from the compilation of field and laboratory data presented in Table 15 of Presser and Luoma, 2006]) and then identified "working values" for the ranges of acceptable selenium concentrations in bird diets and in bird eggs (those shaded in the tables). It is understood that the values will likely be refined during future phases of work (including consideration of site-specific data currently being generated by the Great Salt Lake research effort) and discussion related to establishing a site-specific standard for Great Salt Lake.

A previous draft of this technical memorandum (dated December 8) provided a brief summary of the threshold values that were selected by the Panel during those discussions. For both diet and eggs, the ranges of selenium concentrations selected by the Panel are the lower and upper 95 percent confidence intervals (95% CIs; also referred to as the 5 percent lower confidence limit [LCL] and the 95 percent upper confidence limit [UCL]) for the mean selenium concentration that is associated with a 10 percent reduction (i.e., the 10 percent effect concentration or EC_{10}) in the hatchability of mallard eggs. Those values were reported by Ohlendorf (2003), based on the analysis of data from six laboratory studies (Heinz et al. 1987, 1989; Heinz and Hoffman 1996, 1998; Stanley et al. 1994, 1996). Essentially, there is 95 percent confidence that the mean dietary or egg selenium concentration that causes a 10 percent reduction of egg hatchability is within the identified ranges, which are illustrated in the figures below.

The Panel agreed by consensus that the 95% CIs on mean selenium concentrations in mallard diet and eggs associated with the EC_{10} for egg hatchability would be reasonably protective for birds nesting at the Great Salt Lake, and that the ranges of values represented by the 95% CIs included the concentrations proposed by various Panel members for consideration. Rationale supporting selection of the 95% CIs is provided by the previous technical memorandum (dated November 28) and through discussion at the Panel meeting.

Panel members provided comments on the December 8 draft version of this technical memorandum summarizing threshold values (Attachment B), and Bill Adams provided further data analyses of effect levels in diets and eggs of mallards that are included in this revised draft. Additional considerations and qualifications about the selected dietary and egg concentrations are presented below in the Discussion section.

All concentrations in bird diets or eggs mentioned below are expressed on dry-weight basis.

Selenium in Bird Diets

The dietary selenium EC_{10} for mallards was reported as 4.87 mg/kg, with 95% CIs of 3.56 to 5.74 mg/kg based on reproductive toxicity (egg hatchability) (Ohlendorf 2003). The EC_{10} of 4.87 mg/kg was estimated by fitting a logistic regression model (Figure 1). It should be noted, however, that the mallard studies used a “dry diet” that had about 10 percent moisture. Ohlendorf (2003) used the reported dietary selenium concentrations without adjustment for that moisture content, but an upward adjustment of the values (by 11 percent) would be appropriate to account for the moisture content of the duck diet.

In Adams et al. (2003), hockey-stick regression was used to model relationships between egg selenium concentrations and adverse effects in order to derive toxicity thresholds, such as EC_{10} values. Hockey-stick regression is a model that has been used elsewhere to define a threshold when an underlying background level of response is unrelated to the dose (see Adams comments in Attachment B). Thus, such a model may be relevant to naturally occurring elements that are essential to birds and a wide variety of other organisms and particularly useful for elements such as selenium, which has a narrow range between levels that are essential and those that are toxic to birds so that variance around the inflection point (threshold) in the model is small. As shown in Figure 2 below, a threshold clearly

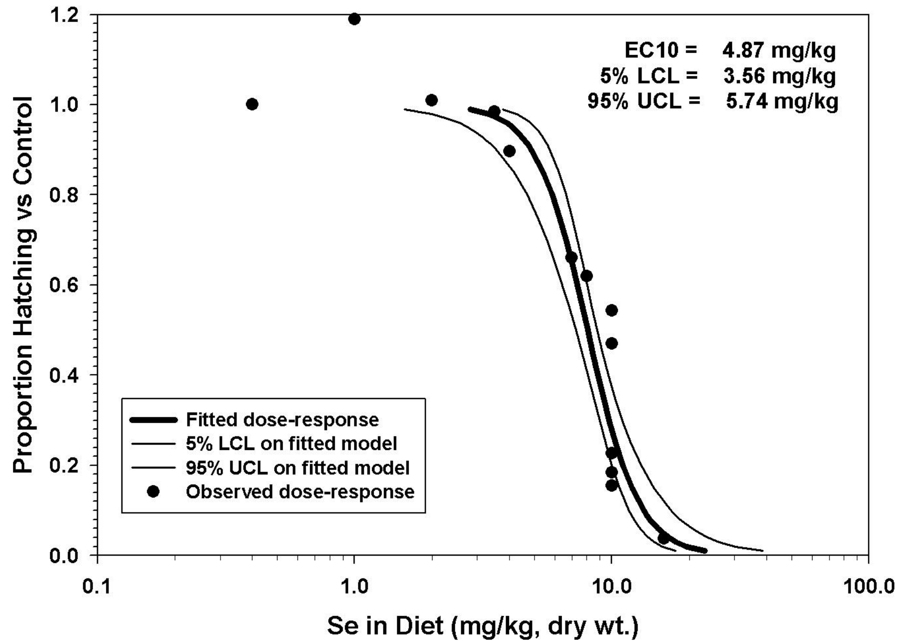

Figure 1. Mallard egg hatchability vs control as a function of selenium concentration in diet.

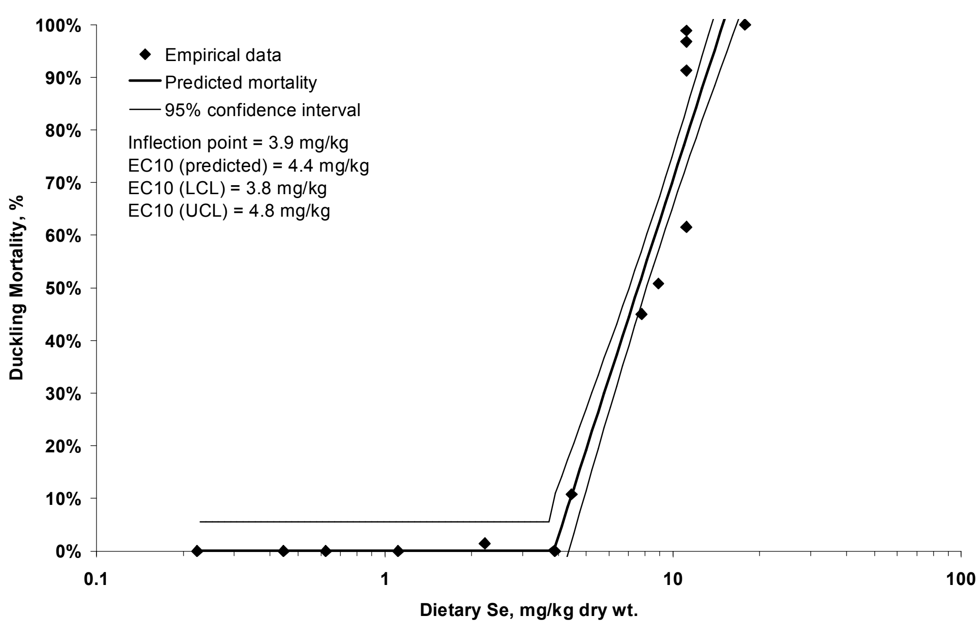

Figure 2. Hockey stick regression of laboratory mallard duckling mortality versus dietary selenium.

appears to exist when dietary selenium is plotted versus duckling mortality (which incorporated the cumulative effects of fertilization success and hatchability). The inflection point occurs at a dietary selenium concentration of 3.9 mg/kg. (The Discussion section below describes uncertainty around the inflection point.) The predicted EC_{10} is 4.4 mg/kg (just slightly above the inflection point) and the 95% CI around the predicted EC_{10} ranges from 3.8 to 4.8 mg/kg. The predicted EC_{10} of 4.4 mg/kg is slightly lower than Ohlendorf's (2003) EC_{10} of 4.9 mg/kg, and the 95% CI is narrower using hockey stick regression than when using logistic regression.

Selenium in Bird Eggs

Similar to the dietary values calculated by Ohlendorf (2003) for reproductive toxicity for mallards, the EC_{10} in eggs was reported as 12.5 mg/kg, with 95% CIs of 6.4 to 16.5 mg/kg (Figure 3). The EC_{10} of 12.5 mg/kg was estimated by fitting a logistic regression model to the results of the six laboratory studies with mallards.

As noted in Table 2, the EC_{10} for duckling mortality, as reported in Adams et al. (2003), ranged from 12 to 16 mg/kg (see Adams comments in Attachment B). These EC_{10} values are based on a synthesis of laboratory studies in which the final endpoint was duckling mortality (the same effects data used in the dietary EC_{10} evaluation with hockey-stick regression above) and the range of EC_{10} values reflects different statistical approaches for analyzing the data. An adaptation from Figure 3 in Adams et al. (2003) is provided below (Figure 4), with the 95% CI included. As shown, the inflection point occurs at an egg selenium concentration of 9.8 mg/kg, with a predicted EC_{10} comparable to that derived by Ohlendorf (2003). (See Discussion for comments concerning uncertainty around the inflection point.) However, the 95% CI using hockey-stick regression is much narrower (9.7 to 13.6 mg/kg) than that derived by Ohlendorf using logistic regression (6.4 to 16.5 mg/kg). Given that there is a clear egg-selenium threshold at which effects begin to be observed, a unimodal model, such as logistic regression, may result in exaggerated confidence intervals, particularly in the tails.

Discussion

Additional discussion is presented below concerning the basis for selection of threshold values, uncertainty surrounding the hockey-stick regression inflection points, hormetic effects of selenium, and other qualifications and points discussed during the Panel meeting in November, as reflected in comments from Panel members (Attachment B).

Basis for Selection of Threshold Values

The Science Panel can choose a scientifically-based threshold value or acceptable "benchmark" concentration based on the consensus confidence limits described by analysis of available data (presented above), but ultimately, a choice of numbers from within the consensus confidence limits for regulatory purposes is not a scientific decision. Choices of a specific number or numbers from within those confidence ranges are philosophical/legal decisions that depend on how precautionary the State of Utah wants to be (a matter of philosophy) and on how much potential for legal liability the State is comfortable with exposing itself to. The key decision the State must make is whether they want to regulate to a "NEC" (no effects concentration, which is not the same as a NOEC [no observed effects

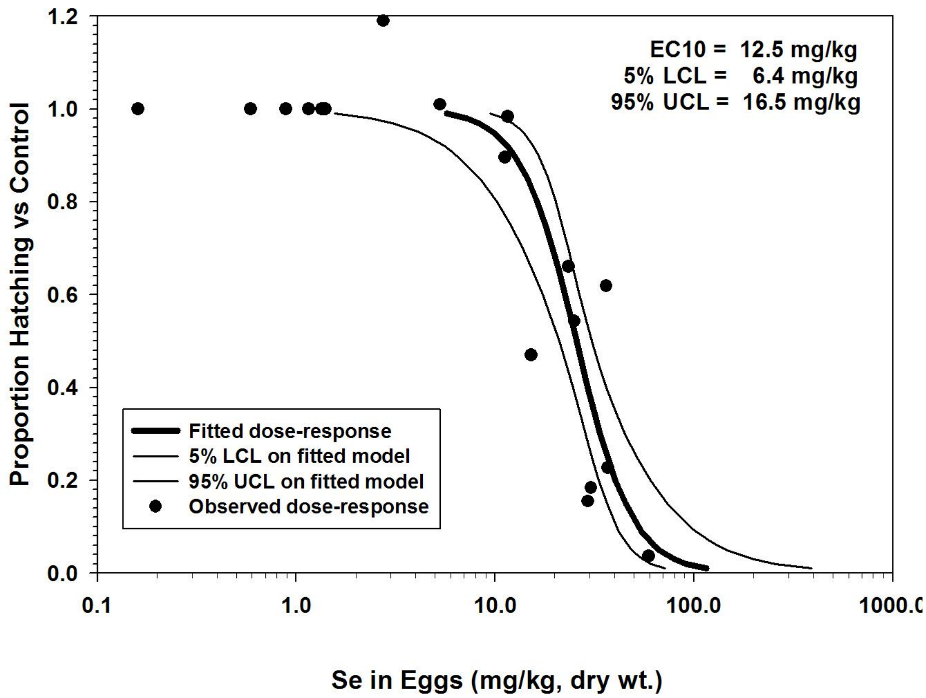

Figure 3. Mallard egg hatchability vs control as a function of selenium concentration in eggs.

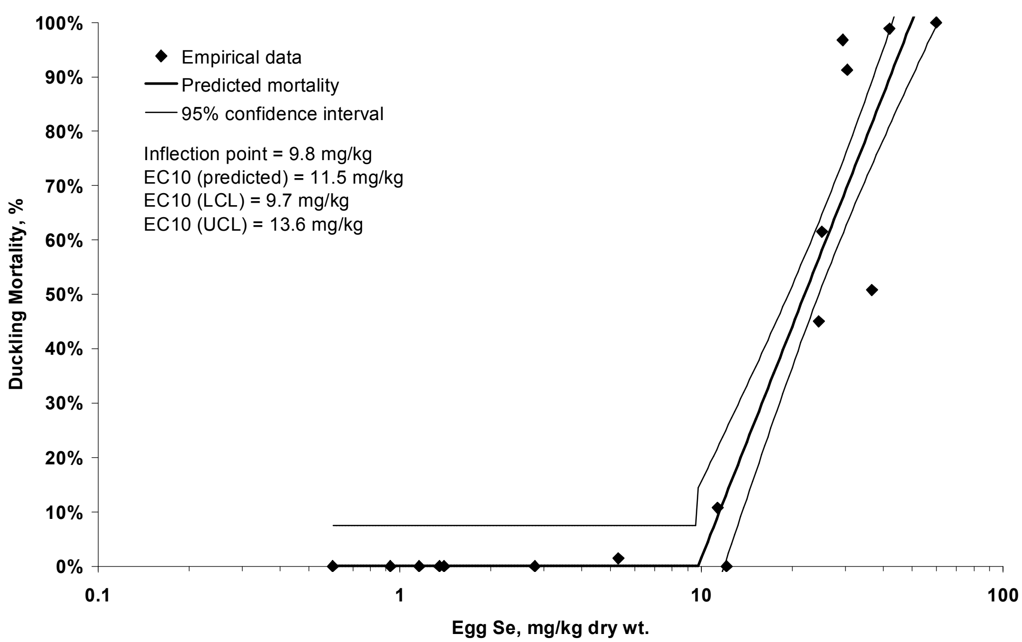

Figure 4. Hockey stick regression of laboratory mallard duckling mortality versus egg selenium.

concentration]) standard or to some version of a "tolerably toxic" standard such as an EC_{10}, an EC_{20}, or an EC_{05}, etc.

Conceptually, a benchmark concentration is defined as the location on the exposure-response curve that is the threshold between absence and presence of a given effect or endpoint (i.e., the threshold between an EC_{00} and an EC_{01} concentration [see: www.epa.gov/ecotox/ecossl/pdf/ecossl_attachment_3-2.pdf; p. A-6]). Benchmark concentrations are estimated as the lower 95 percent confidence boundary on the EC_{10} (see: Meister and Van Den Brink [2000], pp. 114-116 in particular; and USEPA [2000]).

Uncertainty Surrounding the Hockey-Stick Regression Inflection Points

To determine the inflection point between the hockey-stick "blade" and "handle", or any parameter in the model, initial parameter values are input to the software program SPlus® and an iterative technique is used to search for more exact parameter values that will minimize the sum of squared deviations between the observed effects data and effects values predicted by the model. Variance in the estimate of the inflection point value is affected by the spacing of the measured X values as well as the scatter or trend in Y values in the vicinity of the estimated inflection point. If, for example, there are few measured dietary selenium concentrations near the predicted inflection point, the uncertainty in the location of the inflection point will be greater because it will be difficult to determine the exact concentration at which the inflection point occurs (i.e., it could be between two of the measured values). Uncertainty around the predicted Y (EC) values at the predicted inflection point is affected by the number of Y values and the scatter of the Y values at that particular X value (which, when calculating the confidence interval around Y, is assumed to be estimated without error). Thus, both the spacing of the measured X values and the variance in the response variable affects the uncertainty around the inflection point. The tighter spacing and less ambiguous effects response after the inflection point causes the 95% CI around the dietary selenium-based inflection point (3.0 to 4.9 mg/kg) to be narrower than that for the egg selenium-based inflection point (6.4 to 14.9 mg/kg).

However, although there is uncertainty surrounding the inflection point, use of the best estimate of the inflection point results in the best fit of the regression model to the data. In Figure 4, for example, if the inflection point occurred at either end of the 95% CI of egg selenium concentration (6.4 to 14.9 mg/kg dry wt.) one can easily visualize that the fit of the regression to the data points above the inflection point would not pass through the measured values in the same way.

Hormetic Effects of Selenium

Consideration of the hormetic effects of selenium may result in lowering of thresholds (for hormetic substances and endpoints one has to distinguish between valid control responses and hormetic deficiency responses before a valid baseline to compare toxic responses against can be identified). The hormetic bias in the data used for the Ohlendorf (2003) regressions has not yet been fully considered by the Science Panel. If such consideration were to result in changes, those changes could only be in the direction of a downward shifting of the threshold confidence limits. (For example, preliminary unpublished analyses that adjusted for hormetic effects in the mallard data yielded a revised EC_{10} for diet of

4.1 mg/kg, with a 95% CI of 1.3 to 5.8 mg/kg, and a revised EC_{10} for eggs of 9.22 mg/kg, with a 95% CI of 4.11 to 13.07 mg/kg.).

Other Qualifications and Points Discussed

The Panel also discussed the following additional qualifications and points relative to toxicity threshold values:

- Applicability of laboratory data to field situations is not certain (note that field data were retained in compilation of egg-selenium concentrations in Table 2), and it is important to collect site-specific field data on selenium concentrations in bird eggs (e.g., current data gathering effort at the Great Salt Lake).
- Applicability of mallard data to species at Great Salt Lake is uncertain, because relative sensitivity of all species nesting there is not known.
- Threshold values discussed are for the hatchability endpoint (based on diet and avian egg) but non-reproductive adverse effects endpoints (e.g., avian blood endpoint) also may be important. However, interpretive values for selenium in avian blood are not available; although selenium concentrations in blood indicate exposure of the birds, that endpoint is not considered useful for setting a water quality standard.
- Phalaropes are seasonally numerous at the Great Salt Lake and should be added to the list of species to be monitored because they represent species with a feeding rate that is a large percentage of body weight (affecting energy consideration in determining wildlife criterion).

Recommended Next Steps

The issues summarized in this technical memorandum should be discussed/considered further by the Panel, particularly to refine the selection of threshold values for bird diets and eggs with respect to effects documented elsewhere (in field and laboratory studies) and considering the results being developed through research at the Great Salt Lake. In parallel, it will be important to know what level of protectiveness the State and EPA will apply in the development of the site-specific standard for selenium on the Great Salt Lake (i.e., EC_{20}, EC_{10}, EC_{05}, etc.) so that the Science Panel can most effectively make recommendations that can be applied toward that purpose.

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ATTACHMENT A

Tables

TABLE 1
Diet Concentrations

mg/kg	Approach or Site	Effects	Species	Reference(s)
4.87 **(CI 3.56 - 5.74)**	Synthesis of lab Data	Hatchability in mallards (10% effect level/95% confidence boundaries)	Mallard	Ohlendorf 2003
4.4 (CI 3.8 - 4.8)	Synthesis of lab data	EC_{10} for duckling mortality	Mallard	Bill Adams analyses presented in Attachment B
3.85 - 7.7 (diet based on 10% moisture)	Lab	Reduced hatching success in mallards (33% at 7.7 µg/g); reduced growth and weight in hatchlings	Mallard	Stanley et al. 1996
7.7 (diet based on 10% moisture)	Lab	Reduction in number of surviving mallard ducklings produced per female	Mallard	Stanley et al. 1996
8.8 4.4/6.2 (diet based on 10% moisture)	Lab	8.8 - LOAEL, 4.4 - NOAEL, 6.2 - Geometric Mean Reduction (17%) in survival of mallard ducklings; mean decrease (43%) in number of 6-day-old ducklings	Mallard	Heinz et al. 1989
6	Lab	Adverse effect on body condition of male American kestrels	American Kestrels	Yamamoto and Santolo, 2000
7.7 - 8.8 (diet based on 10% moisture)	Lab	Dietary threshold of teratogenic effects in mallards; above upper threshold, rate of deformity rises sharply	Mallard	Stanley et al. 1996
7.7 - 8.8 (diet based on 10% moisture)	Lab	Dietary threshold of mallard duckling mortality (parental exposure)	Mallard	Stanley et al. 1996

Note: Highlighted cells are the threshold values for bird diets identified by consensus of the Science Panel on November 30, 2006.

TABLE 2
Egg Concentrations

mg/kg (dry wt.)	Approach or Site	Effects	Species	Reference(s)
12.5 **(CI 6.4 - 16.5)**	Synthesis of lab data	Hatchability in mallards (10% effect level/95% confidence boundaries)	Mallard	Ohlendorf 2003
10	Synthesis of lab data	NOAEL	Mallard	Adams et al. 2003
12 - 16	Synthesis of lab data	EC_{10} for duckling mortality	Mallard	Adams et al. 2003
9	Synthesis of lab data	Impaired clutch viability (8.2% effects level)	Mallard	Lam et al. 2005
8.2 (or 7.3) (egg based on 73% moisture)	Field	16% depression in egg viability **(*7.3 in paper*)**	Spotted Sandpiper	Harding et al. 2005
6	Synthesis of field data	Threshold (3% effect level) of hatchability	Stilts	Skorupa, 1998; Skorupa, 1999
5.1 (egg based on 78.4% moisture)	Field	15% depression in egg viability	American dipper	Harding et al. 2005

Note: Highlighted cells are the threshold values for bird eggs identified by consensus of the Science Panel on November 30, 2006.

ATTACHMENT B

Comments on December 8, 2006, Draft Technical Memorandum

ATTACHMENT B

Comments on December 8, 2006, Draft Technical Memorandum

Comments of Bill Adams

Following are comments on Harry Ohlendorf's draft technical memorandum to the Great Salt Lake Science Panel entitled *Threshold Values for Selenium in Great Salt Lake: Selections by the Science Panel* (December 8, 2006).

Selenium in Bird Diets

As noted in the draft memorandum, the mallard studies used in Ohlendorf (2003) as the basis for a dietary selenium EC10 in birds was based on a "dry diet" containing about 10% moisture. Although the moisture content of the mallard diet was low, we recommend that standard convention should be used to properly adjust the dietary selenium concentrations to a dry weight basis. The equation for the wet weight-to-dry weight conversion is included in Attachment 1 to this memorandum.

In Adams et al. (2003), hockey-stick regression was used to model relationships between egg selenium concentrations and adverse effects in order to derive toxicity thresholds, such as EC10 values. Hockey-stick regression is a model that has been used to define a threshold when an underlying background level of response is unrelated to the dose. Thus, such a model may be relevant to naturally occurring elements that are essential to birds and a wide variety of other organisms and particularly useful for elements such as selenium, which has a narrow range between levels that are essential and levels that are toxic to birds so that variance around the inflection point (threshold) in the model is small. As shown in Figure 1 below, a threshold clearly appears to exist when dietary selenium is plotted versus duckling mortality (which incorporated the cumulative effects of fertilization success and hatchability). The inflection point occurs at a dietary selenium concentration of 3.9 mg/kg dry wt. (please see discussion at end of comments concerning uncertainty around the inflection point). The predicted EC10 is 4.4 mg/kg dry wt. (just slightly above the inflection point) and the 95% confidence interval around the predicted EC10 ranges from 3.8 to 4.8 mg/kg dry wt. The predicted EC10 of 4.4 mg/kg dry wt. is slightly lower than Harry Ohlendorf's EC10 of 4.9 mg/kg dry wt., but the 95% confidence interval is narrower using hockey stick regression.

Selenium in Bird Eggs

As noted in Table 2 of the draft memorandum, the EC10 for duckling mortality, as reported in Adams et al. (2003), ranged from 12-16 mg/kg dry wt. These EC10 values are based on a synthesis of laboratory studies in which the final endpoint was duckling mortality (the same effects data used in the dietary EC10 evaluation above) and the range of EC10 values reflects different statistical approaches for analyzing the data. An adaptation from Figure 3 in Adams et al. (2003) is provided below, with the 95% confidence interval included. As shown, the inflection point occurs at an egg selenium concentration of 9.8 mg/kg with a predicted EC10 comparable to that derived by Harry Ohlendorf (please see discussion at end of comments concerning uncertainty around the inflection point). However, the 95% confidence interval using hockey stick regression is much narrower (9.7 to 13.6 mg/kg dry wt.) than that derived by Harry using logistic regression (6.4-16.5 mg/kg dry wt.). Given that there is a clear egg selenium threshold at which effects begin to be observed, a unimodal model, such as logistic regression, may result in exaggerated confidence intervals, particularly in the tails.

Uncertainty Surrounding the Hockey-Stick Regression Inflection Points

To determine the inflection point between the hockey-stick “blade” and “handle”, or any parameter in the model, initial parameter values are input to the software program SPlus® and an iterative technique is used to search for more exact parameter values that will minimize the sum of squared deviations between the observed effects data and effects values predicted by the model. Variance in the estimate of the inflection point value is affected by the spacing of the measured X values as well as the scatter or trend in Y values in the vicinity of the estimated inflection point. If, for example, there are few measured dietary selenium concentrations near the predicted inflection point, the uncertainty in the location of the inflection point will be greater because it will be difficult to determine the exact concentration at which the inflection point occurs (i.e., it could be between two of the measured values). Uncertainty around the predicted Y (EC) values at the predicted inflection point is affected by the number of Y values and the scatter of the Y values at that particular X value (which, when calculating the confidence interval around Y, is assumed to be estimated without error). Thus, both the spacing of the measured X values and the variance in the response variable affects the uncertainty around the inflection point. The tighter spacing and less ambiguous effects response after the inflection point causes the 95% confidence interval around the dietary selenium-based inflection point (3.0 to 4.9 mg/kg dry wt.) to be narrower than that for the egg selenium-based inflection point (6.4 to 14.9 mg/kg dry wt.).

However, although there is uncertainty surrounding the inflection point, use of the best estimate of the inflection point results in the best fit of the regression model to the data. In Figure 2, for example, if the inflection point occurred at the either end of the 95% confidence interval of egg selenium concentration (6.4 to 14.9 mg/kg dry wt.) once can easily visualize that the fit of the regression to the data points above the inflection point would not pass through the measured values in the same way.

Figure 1. Hockey stick regression of laboratory mallard duckling mortality versus dietary selenium.

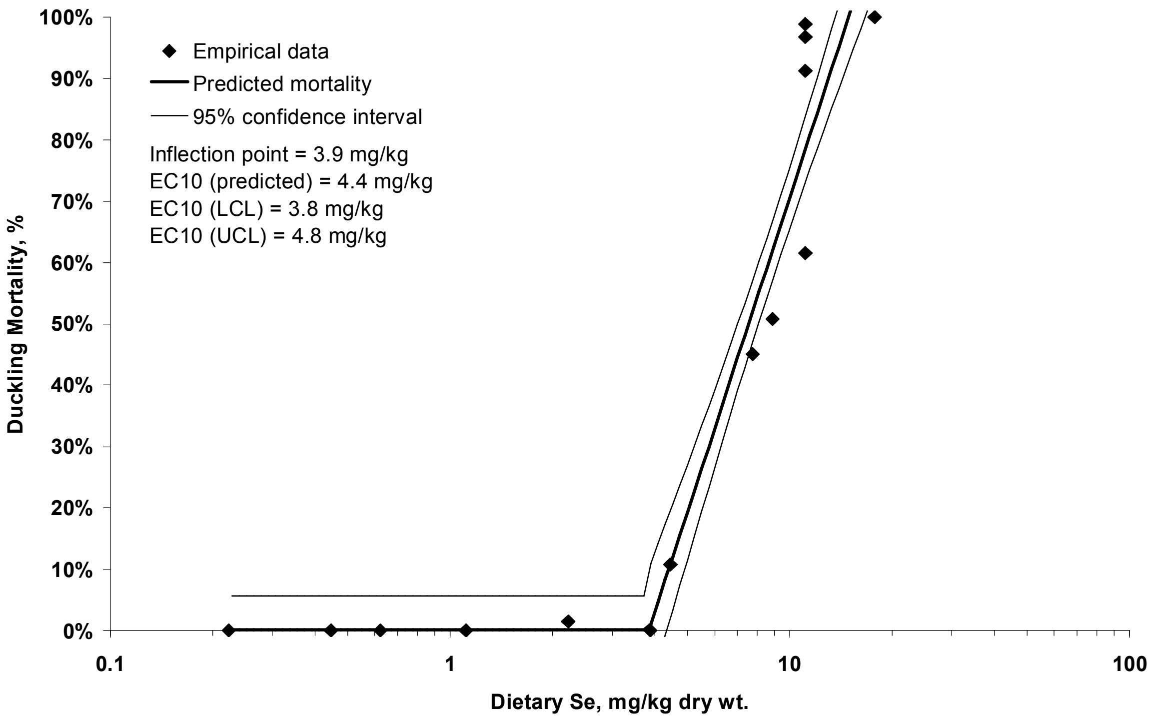
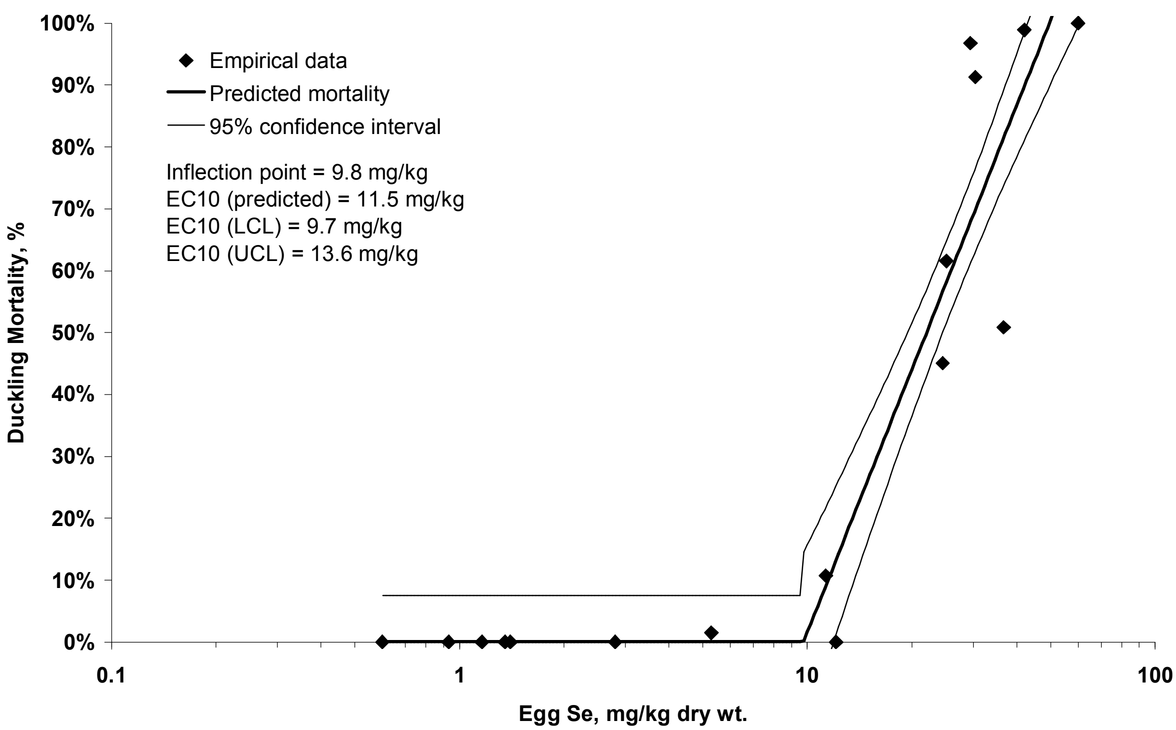


Figure 2. Hockey stick regression of laboratory mallard duckling mortality versus egg selenium.



ATTACHMENT 1

WET WEIGHT-TO DRY WEIGHT CONVERSION FOR DIETARY SELENIUM CONCENTRATIONS IN MALLARD STUDIES

$$\text{Dry Weight Concentration} = \frac{\text{Wet Weight Concentration}}{f_{\text{solids}}}$$

Where: f_{solids} = fraction solids in diet (i.e., 0.9 in a diet containing 10% moisture)

Comments of Anne Fairbrother

I realize that I am late (the last?) on providing comments and feedback on the report you pulled together from our last Salt Lake City meeting on threshold values. I was sort of hoping to see the data from Bill Adams' re-analysis of the dose-response before replying... Absent that, here are my thoughts and comments.

I think you did an appropriate job pulling together what was discussed at the meeting in regard to diet and egg threshold levels. However, the more I look at the data in regard to selenium uptake and effects, the more convinced do I become that we are dealing with a threshold phenomenon, likely because of the essential nature of the element. I do believe that the mean value for the EC10 that was selected for both endpoints is likely to remain pretty much the same regardless of what dose-response model is used, but the standard error about the mean may be different. Likely it will be smaller when using a threshold model since a logistic model tends to spread out the CI's at its tails. So, for now, I am willing to approve the document as a report of what was discussed at the meeting, but not as a final say on what we have agreed to for the EC10 and its confidence intervals.

Comments of Theresa Presser

Suggested additions to threshold discussion write-up of 12/8/06:

1) Page 1: Note that compilation of data for consideration was adapted from Presser and Luoma (2006), table 15.

2) Page 1: Note that in addition to laboratory data, a compilation of field data for egg concentrations was retained.

3) Page 1: Note that any final determination must take into account site-specific data currently being generated by the Great Salt Lake research effort.

4) Page 2 wording: "The panel agreed by consensus that the 95% CIs on mean selenium concentrations in mallard diet and eggs would be reasonably protective for birds nesting at the Great Salt Lake, and the range of values included the concentrations proposed by various panel members for consideration. Rational supporting selection of the 95% CIs is provided by the previous technical memorandum and through discussion at the panel meeting."

    a) Did you mean here the 95% CIs on the mean EC10 for hatchability?

    b) The phrase "would be reasonably protective for birds nesting at the Great Salt Lake" does not adequately convey all parts of the extensive discussion that took place. I did not perceive that a consensus had been reached as to protectiveness, only that a consensus had been reached as to the interpretation of data from mallard lab experiments. Therefore, I suggest incorporating into the wording of a summary statement the following qualifications and points that were discussed at the meeting:

        1) Applicability of lab data to field situations (note retention of compilation of field data in table 2 and current data gathering effort at the Great Salt Lake; points 2 and 3 listed above)

        2) Applicability of mallard data to species at Great Salt Lake (sensitivity issue)

        3) Applicability of hatchability endpoint (diet and avian egg) and non-reproductive adverse effects endpoints (e.g., avian blood endpoint)

        4) Level of protection and precautionary regulation as exemplified by benchmark concentration regulation. Specifically add excerpt from page 8 of 11/28/06 memo as clarification of 95% CI: "Conceptually, a benchmark concentration is defined as the location on the exposure-response curve that is the threshold between absence and presence of a given effect or endpoint, i.e., the threshold between an EC00 and an EC01 concentration (see: www.epa.gov/ecotox/ecossl/pdf/ecossl_attachment_3-2.pdf; p. A-6)….. Benchmark concentrations are estimated as the lower 95% confidence boundary on the EC10 (see: Meister, R., and P.J. Van Den Brink. 2000. The analysis of laboratory toxicity experiments. Pages 99-118 in T. Sparks (ed.), Statistics in Ecotoxicology. John Wiley & Sons, LTD, New York, NY: [pp 114-116 in particular]; and see: USEPA. 2000. Benchmark Dose Technical Guidance

Document. [External Review Draft]. EPA/630/R-00/001. U.S. Environmental Protection Agency, Washington, DC)."

5) Addition of phalarope to list of species to be monitored to represent species with a feeding rate that is a large percentage of body weight (energy consideration in determining wildlife criterion).

6) Potential lowering of thresholds through consideration of hormesis data (for hormetic substances and endpoints one has to distinguish between valid control responses and hormetic deficiency responses before a valid baseline to compare toxic responses against can be identified).

5) References: Add Presser and Luoma, 2006.

6) Table 1:"Bill Adams suggestion" needs to be documented as how his entry differs from entry #1 in table 1.

Comments of Joe Skorupa

In Table 1 I don't believe the science panel wanted the value of 4.87 to be presented in bold type, only the confidence limits (for comparison see Table 2 where I think you have it the way the science panel intended).

Adjusting for 10% moisture would result in an 11% increase in the dietary values, not an upward adjustment of 10% as stated.

I didn't feel like your draft write-up adequately conveyed our (sci. panel's) discussion concerning the fact that, ultimately, a choice of numbers from within the consensus confidence limits is not a scientific decision. That confidence range is as far as science can bring us... choosing a specific number or numbers from within those confidence ranges are philosophical/legal decisions that depend on how precautionary the State of Utah wants to be (a matter of philosophy) and on how much potential for legal liability the State is comfortable with exposing itself to. The key decision the State must make is whether they want to regulate to a "NEC" (no effects concentration... which is not the same as a NOEC) standard or to some version of a "tolerably toxic" standard such as an EC-10, or EC-20, or EC-05 etc.

Finally, I think on the scientific side of things we would be remiss in our duty as experts not to include some discussion indicating that the issue of hormetic bias in the data used for the Ohlendorf (2003) regressions has not yet been fully considered by the science panel (at Bill Adams request to defer it so that he could preview Beckon's SETAC presentation before I presented any of it to the panel... although it seemed to be acceptable to everyone to see Kennecott's U. of Wyoming presentation without any opportunity for anyone other than Bill A. to preview it... seems like a double standard to me), and that if such consideration were to result in changes, those changes could only be in the direction of a downward shifting of the threshold confidence limits.

For example, remember that the analysis that Brad Sample re-ran to adjust for hormetic effects in the mallard data yielded a revised EC-10 for diet of 3.7 ppm ww [4.1 ppm dw] with a 95% confidence interval of 1.15 - 5.18 ppm ww [1.3 - 5.8 ppm dw] and a revised EC-10 for eggs of 9.22 ppm dw with a 95% confidence interval of 4.11 - 13.07 ppm dw.

APPENDIX C

ALLOMETRIC ADJUSTMENT OF DIETARY CONCENTRATIONS

The Appendix explains the approach used to allometrically adjust dietary toxicity threshold for birds. In ecological risk assessments for bids, dose-based toxicity reference values (TRVs) are used to estimate toxic effects. Toxicity reference values are calculated using the following equation:

$$TRV_t = \frac{EPC \times IR \times FC \times AF}{BW}$$

where:

TRV_t = Toxicity reference value for the test species.

EPC = Exposure point concentration; i.e., the dietary concentration of a chemical that produced the toxic effects measured in the toxicity study (mg/kg-dw).

IR = Food ingestion rate (mg/day-dw).

FC = Fraction contacted (unitless; assumed to be 1).

AF = Assimilation fraction (unitless; assumed to be 1).

BW = Body weight (kg).

Toxicity reference values are allometrically adjusted using the following equation (Sample and Arenal 1999):

$$TRV_a = TRV_t \times \left(\frac{BW_t}{BW_a} \right)^{(1-b)}$$

where:

TRV_a = Allometrically adjusted toxicity reference value.

TRV_t = Toxicity reference value for the test species.

BW_t = Body weight of the test species.

BW_a = Body weight of species for which the allometric adjustment is being performed.

b = Allometric scaling factor (unitless).

The $\left(\frac{BW_t}{BW_a}\right)^{(1-b)}$ term in the equation above is also referred to as an "allometric adjustment factor". Combining the above two equations, and eliminating the variables assumed to equal 1, produces the following equation:

$$\frac{EPC_a \times IR_a}{BW_a} = \left[\frac{EPC_t \times IR_t}{BW_t}\right] \times \left(\frac{BW_t}{BW_a}\right)^{(1-b)}$$

By re-arranging the equation above, it is possible to derive an allometrically adjusted dietary concentration protective of specific toxic effect. The re-arranged equation is as follows:

$$EPC_a = \left[\frac{BW_a}{IR_a}\right] \times \left[\frac{EPC_t \times IR_t}{BW_t}\right] \times \left(\frac{BW_t}{BW_a}\right)^{(1-b)}$$

Allometrically adjusted dietary thresholds were calculated for the avian species of concern in the Bay identified by USFWS (2007):

- Bald eagle
- California clapper rail
- Greater scaup
- Lesser scaup
- White-winged scoter
- Surf scoter
- Black scoter

The test species from which the TRVs were allometrically adjusted include:

- Leghorn chicken
- Mallard
- Screech owl

The body weights for all of these birds are provided in Table C-1 below. Ingestion rates for each bird species were calculated from their body weights using the equations provided by Nagy (2001). The ingestion rate equations and calculated ingestion rates are also shown in Table C-1 below.

Table C-1
Body weights and ingestion rates.

Bird species	Body Weight (g)	Ingestion Rate Equation[f]		Ingestion Rate (mg/day-dw)
Bald eagle[a]	5,275	Carnivorous birds	y = 0.849*(x)^0.663	249,311
California clapper rail[a]	346	Marine birds	y = 0.880*(x)^0.658	41,228
Greater scaup[a]	959	Marine birds	y = 0.880*(x)^0.658	80,634
Lesser scaup[a]	663	Marine birds	y = 0.880*(x)^0.658	63,247
White-winged scoter[a]	1,917	Marine birds	y = 0.880*(x)^0.658	127,189
Surf scoter[b]	1,047	Marine birds	y = 0.880*(x)^0.658	85,429
Black scoter[a]	987	Marine birds	y = 0.880*(x)^0.658	82,198
Mallard[c]	1,214	Marine birds	y = 0.880*(x)^0.658	94,167
Chicken[d]	1,800	Galliformes	y = 0.088*(x)^0.891	69,973
Screech Owl[e]	186	Carnivorous birds	y = 0.849*(x)^0.663	27,139

Notes:
a - female body weight from USFWS (2007)
b - female body weight from BNA (2007)
c - body weight is from Heinz et al. (1989) and is average of females at time of sacrifice in all treatments except 16 mg/kg
d - egg laying leghorn chickens average 1,800 g (National Research Council 1994)
e - body weight is from Wiemeyer and Hoffman (1996) and is average of females at time of sacrifice in all treatments
f - from Nagy (2001)

The body weights and ingestion rates from Table C-1were used to calculate allometric adjustment factors and allometrically adjusted dietary threshold concentrations for the species of concern in the Bay (Table C-2).

Table C-2
Allometric adjust factors and allometrically adjusted dietary thresholds.

Bird species	Allometric Adjustment Factor (-) for Dietary Screening Values from				Dietary Screening Value (mg/kg-dw) Adjusted from		
	Mallard	Chicken	Screech Owl		Mallard[a]	Chicken[b]	Screech Owl[c]
Bald eagle	1.34	1.24	1.95		9.7	4.0	104.3
California clapper rail	0.78	0.72	-		2.2	0.9	-
Greater scaup	0.95	0.88	-		3.9	1.6	-
Lesser scaup	0.89	0.82	-		3.2	1.3	-
White-winged scoter	1.10	1.01	-		5.6	2.3	-
Surf scoter	0.97	0.90	-		4.1	1.7	-
Black scoter	0.96	0.89	-		3.9	1.6	-

Notes:
a - EC10 for reduced hatching success from Adams et al. (2003) Ohlendorf (2007) of 4.4 mg/kg-dw
b - effect threshold for reduced hatching success of 3.9 mg/kg-dw from Ort and Latshaw (1978)
c - effect threshold for reduced hatching success of 17.3 mg/kg-dw from Wiemeyer and Hoffman (1996)

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